The Journal of HEREDITY

POLLENLESS PUMPKIN

(SEE PAGE 32)

Mice with Vice Versa Viscera

Pravda Phillipic—Genetics à la Russe

Sex-Linked Canine Hemophilia

A New Mouse Linkage

Fused Finger Joints

Who is the "Dean" of Hybrid-Corn?

VOLUME 39 JANUARY, 1948 NUMBER 1

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

DAVID FAIRCHILD, President — Vice-President, SEWALL WRIGHT

Honorary Vice-Presidents, W. E. CASTLE, LEON J. COLE

C. E. LEIGHTY, Secretary — Treasurer, SAMUEL L. EMSWELLER

Managing Editor, ROBERT C. COOK

Assistant Editor, MORTON STARK

EDITORIAL BOARD

M. DEMEREC — RALPH W. PHILLIPS

H. J. MULLER — R. R. GRAVES — PAUL POPENOE

Members of the Council are ex-officio members of the Executive Board

COUNCIL

O. S. AAMODT, Head Agronomist, Division of Forage Crops and Diseases, Plant Industry Station, Beltsville, Md.

RONALD BAMFORD, Professor of Botany, University of Maryland, College Park, Md.

O. F. COOK, Bionomist (Retired), U. S. Department of Agriculture.

GEORGE M. DARROW, Horticulturist in Charge of Small-Fruit Breeding, Horticultural Field Station, Beltsville, Maryland.

SAMUEL L. EMSWELLER, Principal Ho ticulturist, Division of Fruit and Vegetable Crops and Diseases, Plant Industry Station, Beltsville, Md.

DAVID FAIRCHILD, Agricultural Explorer (Retired), U. S. Department of Agriculture.

W. V. LAMBERT, Research Administrator, Agricultural Research Administration, U. S. Department of Agriculture.

C. E. LEIGHTY, Agronomist (Retired), U. S. Department of Agriculture.

HUGH C. McPHEE, Chief of Animal Husbandry Division, U. S. Department of Agriculture, Washington, D. C.

FREDERICK H. OSBORN, Deputy United States Representative on the United Nations Atomic Energy Commission, and on the United Nations Commission for Conventional A maments.

MARCUS M. RHOADES, Professor of Botany, Columbia University, New York City.

SEWALL WRIGHT, Professor of Zoology, University of Chicago.

ADVISORY COMMITTEE

W. S. ANDERSON, Genetics Department, University of Kentucky, Lexington, Kentucky.

E. B. BABCOCK, Genetics Department, University of California, Berkeley, California.

E. A. BESSEY, Professor of Botany, Michigan State College, Lansing, Michigan.

A. F. BLAKESLEE, Professor of Botany, Smith College, Northampton, Mass.

W. E. BRYAN, Professor of Plant Breeding, University of Arizona, Tucson, Arizona.

RUSSELL W. DUCK, Editor, *The Rural New Yorker*. New York City.

C. P. GILLETTE, President, The Genetic Foundation, Fort Collins, Colorado.

C. M. GOETHE, Sacramento, California.

HARRISON B. HUNT, Professor of Zoology, Michigan State College, East Lansing.

D. F. JONES, Plant Breeder, Connecticut Agricultural Experiment Station, New Haven, Conn.

M. A. JULL, Professor of Poultry Husbandry, University of Maryland, College Park, Md.

F. B. LINFIELD, Director, Montana Experiment Station, Bozeman, Montana.

C. C. LITTLE, Director, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.

JAY L. LUSH, Professor in Animal Husbandry, Iowa State College, Ames, Iowa.

H. H. NEWMAN, Emeritus Professor of Zoology, University of Chicago, (Clearwater, Fla.).

T. S. PALMER, Expert in Game Conservation, U. S. Dept. of Agriculture, Washington, D. C.

JOHN H. PARKER, Director, Midwest Barley Improvement Association, Milwaukee, Wisconsin.

JOHN W. SCOTT, Professor of Zoology, University of Wyoming, Laramie, Wyoming.

A. D. SHAMEL, Physiologist, U. S. Department of Agriculture, Riverside, California.

GEORGE H. SHULL, Professor of Biology, Princeton University, Princeton, New Jersey.

EDMUND W. SINNOTT, Professor of Botany, Yale University, New Haven, Conn.

W. W. SMITH, Professor of Animal Husbandry, Purdue University, Lafayette, Indiana.

A. B. STOUT, Director of Laboratories, New York Botanical Gardens, New York City.

W. T. SWINGLE, Plant Physiologist (Retired). U. S. Department of Agriculture.

W. H. TOMHAVE, American Aberdeen-Angus Breeders' Association, Chicago, Ill.

E. N. WENTWORTH, Armours' Bureau of Agr'l Research and Economics, Chicago, Illinois.

P. W. WHITING, Zoological Laboratory, University of Pennsylvania, Philadelphia, Pa.

The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore 11, Md., and 1507 M St., N. W., Washington 5, D. C. Address all communications regarding editorial matters to 1507 M. St., N. W., Washington, D. C. Communications regarding the business management of THE JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to 1507 M St., N. W., Washington 5, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103. Act of October 3, 1917 authorized December 29, 1918.

Contents copyrighted 1948 by the American Genetic Association. Unauthorized reproduction of material from THE JOURNAL not permitted. The title "Journal of Heredity" is registered as a trade-mark in the U. S. Patent Office.

$4.00 a Year. 40c a Copy. Prices of back numbers on application.

The

Journal of Heredity

Vol. XXXIX **JANUARY, 1948** **No. 1**

CONTENTS

SEX-LINKED HEMOPHILIA IN DOGS
By F. B. Hutt, C. G. Rickard, R. A. Field 3
"THE TRUTH" ABOUT GENETICS (The complete text of the recent *Pravda* denunciation. Translated from the Russian.)
By I. Laptev 18
FURTHER DATA ON SYMPHALANGISM
By Arthur G. Steinberg and Earle L. Reynolds 23
LINKAGE OF WHITE AND WAVED-1
By Helen Bunker and G. D. Snell 28
INHERITED VISCERAL INVERSION IN MICE
By Joseph A. Tihen, Donald R. Charles and Theodore O. Sippel 29
A MALE AND FEMALE STERILE VARIANT IN CUCURBITA PEPO
By Lawrence C. Curtis and John Scarchuk 32

Reviews and Notes

ALBINISM IN THE AUSTRIAN PINE 9
BIRTHS AT NEW HIGH IN 1947 10
THE LAY OF THE CORN-HUCKSTER (Rev. of Crabb: *The Hybrid-Corn Makers*)
By Frederick D. Richey 11
A TETRAPLOID SPORT OF MCINTOSH APPLES
By Haig Dermen and George Darrow 17
NEW SOVIET OUSTER CAUSED BY ARTICLE 21
UNAMERICANA 22
DENTAL CARIES, NUTRITION AND HEREDITY 28
BREEDING RED CLOVER (Rev.) 31

An Introduction to Human Heredity and Eugenics:

HOW HEREDITY BUILDS OUR LIVES

BY ROBERT C. COOK AND BARBARA STODDARD BURKS

Illustrated with drawings by Clyde E. Keeler and photographs and charts from the *Journal of Heredity*. This booklet offers the student an informal introduction to heredity, environment, and their interactions.

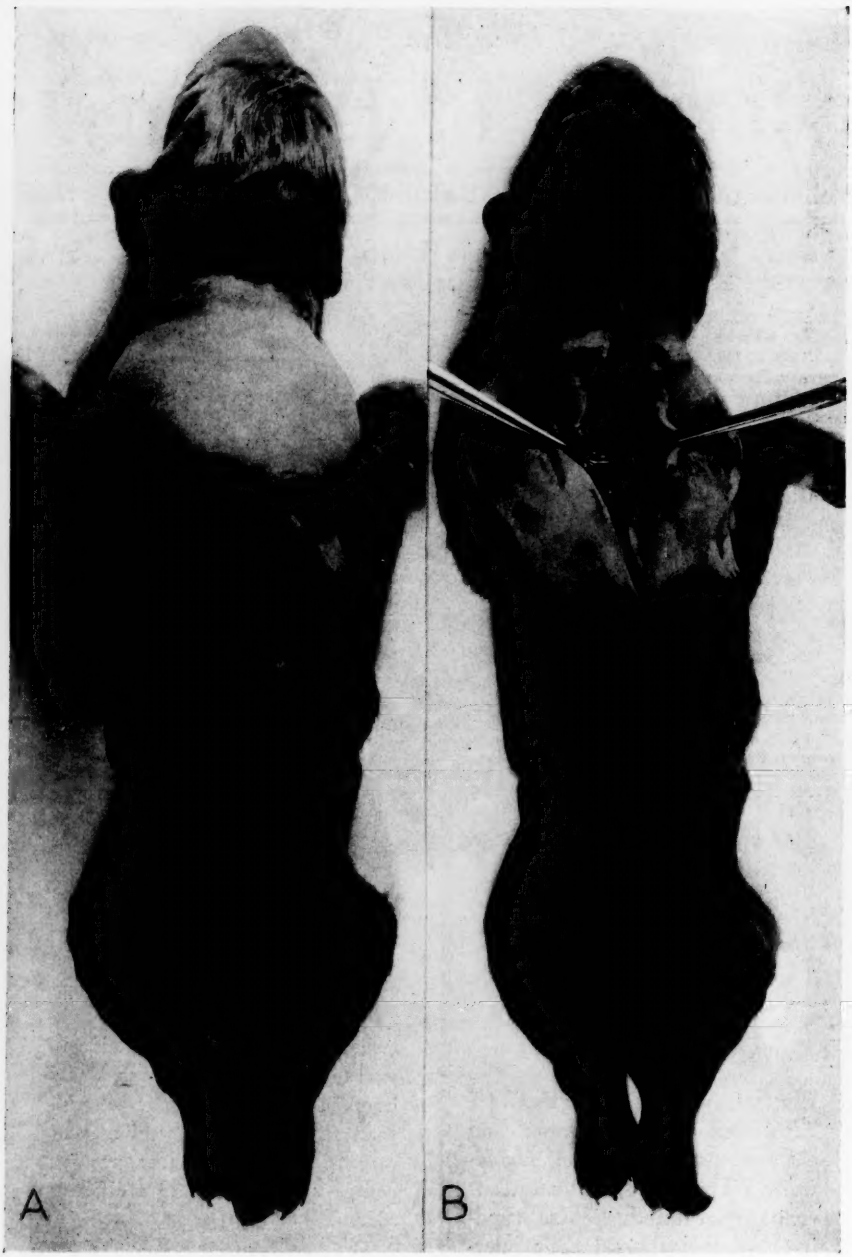
64 pages; 27 figures; 75c per copy—$5.00 per dozen

AMERICAN GENETIC ASSOCIATION
1507 M St., N. W., Washington 5, D. C.

PLEASE

IF YOU ARE CHANGING YOUR ADDRESS, please let us know, if possible, four weeks in advance, giving old as well as new address, and adding zone number to meet postal requirements. If you are unable to notify us, so as to affect succeeding issues, kindly leave instructions and postage with your postmaster for forwarding your copy. Second class matter requires postage when remailed to a different address.

JOURNAL OF HEREDITY
1507 M Street, N. W.
Washington 5, D. C.



DEATH ON THE X CHROMOSOME

Figure 1

Hemophilic puppy, one week old, that died a few hours after the owner noticed the large swelling in the region of the shoulders. The hair was shaved off after death. *Right:* The underlying hematoma was revealed when the skin was cut.

SEX-LINKED HEMOPHILIA IN DOGS

F. B. Hutt, C. G. Rickard, and R. A. Field*
Cornell University, Ithaca, New York

In September, 1946, there was brought to the small-animal clinic of the New York State Veterinary College here a puppy afflicted with complete paralysis of the posterior half of the body. Subsequent study of this dog and of other affected puppies in the same kennel showed that the cause of their trouble was hemophilia of a type apparently identical with the well-known hemophilia of man. Moreover, as genetic evidence accumulated it became clear that the hemophilia in these dogs was caused, *mirable dictu*, by a recessive sex-linked gene just as is hemophilia in man. The details follow.

Description

The most typical symptom by which the owner could identify the hemophilic puppies was a lameness which developed sometime between six weeks and three months of age. This was readily recognized, partly because the lame puppies cried with pain and favored the affected leg, but also because there was usually a marked swelling of one or more joints. Because of these symptoms, the condition had been diagnosed by some veterinarians as rickets. Dissection revealed that the swellings (and resultant lameness) were caused by hemorrhage into the joints, a condition which is also typical of hemophilia in man. The effects of these hemorrhages upon the joints and bones (in man) have been fully described and illustrated by Birch[3] and others.

The hemophilic puppies were also characterized by large swellings that formed under the skin in various regions. These usually appeared suddenly and regressed slowly, but some eventually disappeared. Examination showed that in each case the underlying mass was a hematoma. The hemorrhages giving rise to these hematomata could not be traced to known injuries but they apparently resulted from minor bruises sustained during the ordinary play of the puppies. They occurred in various regions of the body—on the head, in the neck and shoulder region, on the back, and on the limbs. A typical example is shown in Figure 1.

The half-paralyzed puppy mentioned in the opening paragraph was found to have a hematoma in the spinal cord at the region of the emergence of the fourth and fifth lumbar nerves. This had caused considerable destruction of the cord and left only a small ring of tissue around the periphery of the hematoma.

In some cases the owner had been able to recognize the hemophilic puppies at birth by persistent bleeding from the umbilical cord. For some of these it was necessary to ligate the cord at that time to prevent the hemorrhage from causing death.

Viability

So far as one may judge from experience with the dogs concerned in this report, it is apparently more difficult to raise hemophilic puppies than hemophilic boys. Since the time when the owner recognized the disease as a specific clinical entity, no less than 17 hemophilic puppies have been born, but not one of these has been raised to sexual maturity. Two lived to about seven months of age. In most cases the puppies were reduced to such a pitiable state, sometimes partially paralyzed, that they had to be destroyed.

In man, Andreassen[2] found the mean age at death for 93 hemophilics to be 18 years. From this and the average life span of contemporary normal males, he calculated that the duration of life for Danish hemophilics is about one-third that of normal males. Actual duration

*Of the N. Y. State College of Agriculture (F.B.H.) and the N. Y. State Veterinary College (C.G.R. and R.A.F.).

of life cannot be given for these hemophilic puppies because many of them were destroyed as soon as their condition became hopeless. It seems clear from the data given above, however, that hemophilia decreases the life span in the dog more severely than in man. This is to be expected because of the limitations in the care that can be provided for the dogs.

One puppy bled profusely from the gum following eruption of a tooth. This hemorrhage could not be stopped by the use of hemostatic agents or collodion, and the dog was eventually destroyed. Another ruptured a blood vessel supplying a limb when picked up by the owner and the hemorrhage proved fatal.

Tests for Hemophilia

A number of different tests were utilized to determine whether or not the abnormality in these dogs was identical with the hemophilia of man. These have been reported with full details by Field, Rickard, and Hutt[7], and are merely summarized in this paper.

Coagulation Time

The outstanding feature of hemophilia is the prolongation of the time required for clotting. This was measured by the method of Lee and White[9]. Blood was aspirated from the cephalic vein and one cc. put in each of five test tubes having an inside diameter of eight mm. These tubes were held at 37° C. in a water bath and tilted at half-minute intervals. The period from the drawing of the sample to the time when the tube can be inverted without disturbing the clot is the coagulation time. For each test, five tubes were run simultaneously and the average was taken as the final figure. In nine such tests made with four hemophilic dogs, the clotting time ranged from 22.3 to 39.6 minutes. In marked contrast to this, samples of blood from 17 related normal dogs showed clotting times varying only from 2.8 to 6.3 minutes.

Duke's Test of Capillary Bleeding

It is usual for writers on hemophilia to state in one part of their paper that the disease is characterized by prolonged bleeding, and in another that "the bleeding time is normal." The confusion inevitably caused by these contradictory statements is dispelled if it is made clear that the "bleeding time" refers only to capillary hemorrhage. In such cases a comparatively small amount of blood comes in contact with a relatively large amount of injured tissue cells. These liberate enough thromboplastin—the element deficient in hemophilia—to effect normal clotting even in hemophilic persons.

In making such tests (method of Duke[6]) incisions were made in the muccous membrane of the lip just deep enough to cause capillary hemorrhage. In nine tests with three hemophilic puppies, bleeding stopped in 36 to 96 seconds. Since these figures were almost identical with the range of 38 to 120 seconds determined for a normal dog, it is clear that "the bleeding time was normal" as in the hemophilia of man.

Quick's Test for Hemophilia

In normal blood drawn from a blood vessel, clotting starts when the distintegrating blood platelets have released enough thromboplastin for that process. It is believed by some investigators (Minot and Lee[12], Quick[16], and others) that in hemophilic blood the platelets are unusually stable and hence do not release thromboplastin as rapidly as do those of normal blood. Lysis of normal platelets is accelerated by the addition of an oxalate and then a soluble calcium salt. The fact that the platelets of hemophilic blood are comparatively resistant to this treatment has been utilized by Quick[16] in devising a specific test for hemophilia. In his procedure an oxalated sample is evenly divided, and half of it is centrifuged for five minutes at 1,000 r.p.m. This is not enough to throw down the platelets, which remain in the supernatant plasma, a potential source of thromboplastin. The other half of the sample is centrifuged at 3,000

r.p.m., a speed that does precipitate the platelets, and thus leaves the supernatant plasma with only what little thromboplastin it may contain from the platelets that were lysed before centrifugation. After the addition of calcium to the two samples the coagulation time is measured for each. In normal blood, the two samples differ little, if at all, because lysis of the platelets has released plenty of thromboplastin in both before centrifugation. With hemophilic blood, however, clotting of the plasma centrifuged at 3,000 r.p.m. is significantly slower than that of the plasma centrifuged at 1,000 r.p.m.

Quick's test was tried at different times on three samples of blood from each of two hemophilic puppies, and with two samples from a third. All of these tests were positive. The average clotting time was almost twice as long for the plasma centrifuged at 3,000 r.p.m. as for that centrifuged at 1,000 r.p.m. (Table I). As controls, samples of blood from three normal litter sisters of the hemophilic puppies were given the same test. With these, the two speeds of centrifugation caused little or no difference in clotting time, thus demonstrating a negative test for hemophilia.

Other Tests

It is generally stated that the time required for clot retraction, or separation of the serum from the clot is normal in hemophilia, but prolonged in *purpura hemorrhagica*. With these hemophilic dogs, the clot-retraction time was 30 to 60 minutes, which is within the normal range. Andreassen[2] has devised a special apparatus with which he has demonstrated that in hemophilic blood retraction begins later and is finished later than in normal blood, although the clot is normal. This method came to our attention after these studies were completed, and was therefore not tried.

TABLE I.—Average clotting time in seconds for oxalated, centrifuged and recalcified blood of hemophilic and normal dogs. (Quick's test.)

SOURCE OF BLOOD	SPEED OF CENTRIFUGATION	
	1000 r.p.m.	3000 r.p.m.
Hemophilic males		
R. L.	298	520
L. L.	280	610
E. M.	375	780
Their normal litter sisters		
R. E.	90	100
L. E.	95	135
B. E.	120	120

Purpura hemorrhagica is also characterized by a deficiency of platelets. In the hemophilic dogs these cells, at 240,000 to 895,000 per cubic millimeter, were normal according to the range given by Wirth[19] for the dog. Other tests showed that the hemophilic blood was not deficient in prothrombin or fibrinogen.

Sex-Linked Inheritance

By the time these cases came to the attention of the authors, there had been in the one kennel four litters containing hemophilic puppies. All of these, to the number of 11, were males. This suggested that the condition was sex-linked as in man. The results in additional litters have shown this to be true. With the exception of one litter (in which three males died at a few days of age before they could be classified as hemophilic or not) the records of all litters to date from all known carrier females are given in Table II.

Since this hemophilia is caused by a sex-linked recessive gene, the expectation in these litters from heterozygous ♀♀ × normal ♂♂ is that half the male puppies will be normal, half hemo-

TABLE II.—Data for litters from four females heterozygous for hemophilia.*

DAM AND SIRE (Initials only)	PROGENY				
	MALES		FEMALES		
	N. number	H. number	N. number	H. number	Proven carriers number
♀T × ♂P	3	0	5	0	
" "	8	0	5	0	1–B
" "	1	2	7	0	2–N & L
" "	1	2	3	0	
" × ♂C.R.	2	1	2	0	
♀B × ♂M	3	3	1	0	
♀N × ♂S	3	4	4	0	
♀L × ♂P	2	5	5	0	
Totals, observed:	23	17	32	0	
Totals, expected	20	20	32	0	

*N. stands for normal, H. stands for hemophilic.

philic, and all of the females normal. Agreement between expectation and the numbers actually observed was quite good. The critical point is that not one of the female puppies was hemophilic. The excess of males (40 ♂ ♂ :32 ♀ ♀) is normal in this species. Among the males, the ratio of 23 normal:17 hemophilic means a deviation of 3 from the expectation of 20 in each class, but this is not significant ($\chi^2 = 0.9$, n = 1, p = .3 — .5).

The symbol *h* is suggested for this sex-linked, recessive character. Normal males are *H-*, hemophilics, *h-*; normal females, *HH*; and heterozygous females, *Hh*.

The excess of normal males came in the first two litters from the bitch, T. These contained 11 males, none of which was hemophilic. Several puppies of the first litter, including two males, died of distemper at six to eight months of age, but were not recorded as showing any of the abnormalities later recognized as symptoms of hemophilia. Although the eight males of T's second litter were apparently all normal, one of their sisters, B, proved to be a carrier. If T had inherited the gene for hemophilia, half of her ova should have carried it, and the probability of getting by chance 11 successive normal males would be $(\frac{1}{2})^{11}$, or only 1 in 2,048.

It seems more probable, therefore, that the mutation occurred in the female, T, after formation of the ovaries had begun, and that it was not present in all her oöcytes. In such a case, a deficiency of hemophilic offspring would be possible in the offspring of T, but not in the progeny of her heterozygous daughters. This is exactly what occurred, for the litters of B, N and L show a ratio in males of eight normal: 12 hemophilic.

The female B had a second litter that contained six ♂ ♂ and three ♀ ♀. Three of these died when two days old. On examination two were apparently normal and the third possibly hemophilic, but because of the uncertainty of diagnosis at that age, the whole litter is excluded from Table II.

Four daughters of the bitch T were bred. Three of these, B. N, and L, proved to be carriers, and the fourth, with five normal puppies of each sex, was probably not.

The male, P, that sired four hemophilic puppies born to T, also had by other females a total of 43 (known) offspring including 24 males, none hemophilic. The records of F and T thus conform to the expectation that normal males will not transmit hemophilia, while apparently normal females will do so if they carry the causative gene.

Hemophilia in Other Dogs

We have found only two previous reports of hemophilia in dogs. Although neither of these included any tests of the blood, it seems probable that both referred to mutations identical with that considered in this paper.

McKinna[10] observed in the offspring of an Aberdeen terrier bitch three males with symptoms of hemophilia, one of which died from a large abdominal hematoma at 21 months of age. A fourth brother was said to have the same condition. From the Dutch East Indies, Merkens[11] reported hemophilia occurring in greyhounds, with one female producing in four litters eight hemophilic males, seven normal males, and three normal females. The afflicted males apparently all died when comparatively young. In Denmark, Andreassen[2] was told of a condition believed to be hemophilia that occurred in two brother Scotch terriers. Since one of these died of bleeding at three years and the other was still alive at nine years, it seems possible that some other hemorrhagic condition was present. Otherwise, it would appear (as McKinna's report also suggests) that Scottish breeds of dogs are unusually hardy, a situation which, if true, should surprise no one.

The actual frequency of canine hemophilia is undoubtedly greater than one would think from the paucity of records and of the veterinary literature concerning it. One reason for its being little known may be that owners of pure-bred dogs are reluctant to incur publicity

about any hereditary defects in their stock. While any gene for hemophilia in dogs is likely to be eliminated in a few generations, as usually occurs in man, new mutations are to be expected. According to Haldane's[8] latest estimate, the frequency of mutation to hemophilia in man is 3.6×10^{-5}, or about once in 28,000 chromosomes per generation. One can only speculate whether or not a similar high mutation rate applies to hemophilia in the dog.

Hemophilia in Other Species

So far as the authors can determine, hemophilia is known only in man, dogs, and swine. The hemophilic pigs described by Bogart and Muhrer[4] had all the usual clinical manifestations of the condition as they are known in man and here described in dogs. Moreover, tests of the blood by Muhrer, Hogan, and Bogart[15] showed a prolonged coagulation time for whole blood, a "normal bleeding time" by Duke's test and positive Quick's test with oxalated plasma centrifuged at low and high speeds. Blood platelets were unusually resistant to hypotonic salt solutions.[14] Altogether, these characteristics indicate a condition indistinguishable from the hemophilia of the other two species.

The following comparisons show that hemophilia was more serious in the dogs here considered than in the pigs:

	Dogs	*Swine*
Survival to sexual maturity	No	Yes
Maximum duration of life	7 mos.	3 years
Coagulation, normal blood, minutes	Ave. 4.6	Ave. 6.2
Coagulation, hemophilics, minutes	22-39	Ave. 14

Such differences in the severity of a defect are to be expected among species and among breeds within one species. They do not necessarily indicate any difference in the nature of the responsible mutation, but are equally attributable to the influence of modifying genes. There may also be in any species different alleles causing different degrees of severity as Haldane has postulated for hemophilia in man.

In these pigs, typical hemophilia was inherited as an autosomal recessive character. This should interest compilers of criteria for the differential diagnosis of hemorrhagic diseases, most of whom list sex-linked inheritance as one of the essential indications of hemophilia.

While the mode of inheritance may be helpful in diagnosis, it is not necessarily always the same for one disease. The statement that "hemophilia is a disease of the male" does not hold for swine and may not for other species, even for man. It is well to remember that *retinitis pigmentosa* may be sex-linked in some families, autosomal in others, and (in the former) either dominant or recessive.

Significance

The following points seem of interest.

(1) Any positive evidence of a sex-linked gene in mammals, such as the one herein reported, will be welcomed by geneticists because the comparative rarity of such mutations has been somewhat of a mystery. In man, a dozen or more such genes are known. Sex-linked genes are known in the canary, pigeon, turkey, budgerigar, and fowl, the last-named having at least ten that have been recognized to date. In contrast to this, although a systematic search of the literature has not been made, the only well-substantiated case of a sex-linked gene in lower mammals known to the authors is that for yellow and tortoiseshell color in the cat. The mammal best explored genetically (other than man) is the mouse, but, of 57 genes known in that species up to 1945, none is sex-linked.[17]

(2) It is surprising, to say the least, that with 24 different chromosomes available in man and 39 (both haploid numbers) in the dog (Minouchi[13], Ahmed[1]) a mutation preventing normal clotting of the blood should occur in the sex chromosome in both species. One would be tempted to speculate about possible relationships between genes, enzymes, or other determiners of sex and those affecting blood formation, were it not that a mutation with apparently identical effects on the blood occurred in an autosome in the pig.

(3) Dogs with hemophilia can be utilized to learn more about hemophilia in man. In fact, the mutation could not have occurred in a more useful species if it had been made to order. Basic physiology is probably as well known in the dog as in man, and there should be a solid background of facts against which the abnormality can be studied. Stock for this purpose has been obtained by Dr. K. M. Brinkhous, of the Medical College of the University of North Carolina.

(4) In the extensive literature on hemophilia, there is much debate over the possibility that sex-linked hemophilia may occur in women, and the problem of the viability of the homozygote. These questions could be answered experimentally if some of the hemophilic males could be raised to breeding age and mated with heterozygous females. Artificial insemination may prove helpful for this purpose. Although none of 17 such males has been reared to maturity, that may yet be accomplished with special care. Another possibility, suggested by the reports of McKinna and Andreassen, is that the viability of short-legged breeds like Scotch terriers may be reduced by hemophilia less than that of longer-legged breeds like Merkens' greyhounds and the dogs considered in this paper. If so, it may prove feasible to raise hemophilic puppies to maturity by transferring the gene to some short-legged stock.

(5) This mutation is a lethal character, at least in the breed of dogs concerned in this paper. Some of the relatives of the hemophilics were highly prized—and highly priced—because of their own winnings on the show bench, and others had pedigrees of similarly illustrious ancestors. The mutation has obviously caused considerable monetary loss. Once the genetic nature of the condition became clear, the owners refrained from selling any female that could have carried the gene and appropriate steps were taken to eliminate it from the stock. In spite of this, there is some risk, as when any undesirable mutation occurs in any purebred stock, that rumors concerning it may imperil the reputation of the whole kennel. For that reason, and at the request of the owner, the name of the breed concerned is withheld from publication.

Fortunately for this kennel, expurgation of an undesirable mutation is less difficult and less costly with a sex-linked gene than with an autosomal one. Normal brothers of hemophilics cannot carry the gene and can safely be used for breeding. Of their sisters, half are likely to be *Hh*, half *HH*. If these females were particularly needed for further breeding, their genotypes could be tested by mating them with any male, but two or three litters might be necessary to prove which are *HH*. The safest and easiest plan would be to avoid breeding from daughters of any female known to carry the mutation.

Another possibility is that the normal condition may be incompletely dominant in heterozygotes, and that this may prove detectable by some special test. For this purpose the Lee-White technique was ineffective, for 3 proven *Hh* females had coagulation times of 4.8, 3.3, and 2.2 minutes, which did not differ from the figures for normal males. It was learned later that both Studt[18] and Andreassen[2] had found the clotting time to be slightly prolonged in women heterozygous for hemophilia, when it was measured by the method of Bürker[5]. By that time, only one known heterozygote was available here.

Summary

Beginning usually between 6 and 13 weeks of age, hemophilic puppies showed lameness, swelling of joints from hemorrhages, and large subcutaneous swellings elsewhere from the same cause. Some bled to death from slight injuries. Others became partially paralyzed and had to be destroyed. None of the hemophilic dogs lived longer than seven months.

The blood was characterized by an abnormally long time for coagulation and increased stability of platelets as measured by Quick's test for hemophilia.

Capillary bleeding time (Duke's method) was normal, as were number of platelets and amounts of prothrombin and fibrinogen.

All of the hemophilic dogs were males. In eight litters from heterozygous females there were 23 normal males, 17 hemophilic males, and 32 females, all normal. The character is clearly caused by a sex-linked recessive mutation as in man. The significance of this is briefly discussed.

Literature Cited

1. Ahmed, I. A. *Proc. Roy Soc. Edinburgh* 61, B. 107-118. 1941.
2. Andreassen, M. *Opera ex Domo Biol. Hered. Humanae Univ. Hafniensia* 6:1-168. 1943.
3. Birch, C. L. *Illinois Med. Dent. Monographs,* I, No. 4:1-151. 1937.
4. Bogart, R., and M. E. Muhrer. *J. Hered.* 33:59-64. 1942.
5. Bürker, K. *Pflüger's Arch. f. d. ges. Physiol.* 149:318-326. 1913.
6. Duke, W. W. *J. Amer. Med. Assoc.* 55:1185-1192. 1910.
7. Field, R. A., C. G. Rickard, and F. B. Hutt. *Cornell Vet.* 36:285-300. 1946.
8. Haldane, J. B. S. *Ann. Eugen.* 13: 262-271. 1947.
9. Lee, R. I., and P. D. White. *Amer. J. Med. Sci.* 145:495-503. 1913.
10. McKinna, W. R. *The Vet. Journ.* 92: 370-371. 1936.
11. Merkens, J. *Ned. Ind. Bladen v. Diergeneesk. u. Dierent.* 50:149-151. 1938.
12. Minot, C. R., and R. I. Lee. *Arch. Intern. Med.* 18:474-495. 1916.
13. Minouchi, O. *Jap. J. Zool.* 1:255-268. 1928.
14. Muhrer, M. E., R. Bogart, and A. G. Hogan. *Amer. J. Physiol.* 141:449-453. 1944.
15. Muhrer, M. E., A. G. Hogan and R. Bogart. *Amer. J. Physiol.* 136:355-359. 1943.
16. Quick, A. J. *Amer. J. Med. Sci.* 201: 469-474. 1941.
17. Staff, Roscoe B. Jackson Memorial Laboratory. *J. Hered.* 36:271-273. 1945.
18. Studt, H. *Arch. f. Rass.-u. Gesell.-Biol.* 31:214-245. 1937.
19. Wirth, D. *Grundlagen einer klinische Hämatologie der Haustiere.* Berlin. Urban and Schwarzenberg, 1931.

◆

ALBINISM IN THE AUSTRIAN PINE

EARLY in 1947 a collection of 25 unopened cones was made from an isolated specimen of Austrian pine, *Pinus nigra* var. *austriaca.* This species is of common occurrence on the farms of southern Wisconsin outside the range of native pines. Specimens, often solitary, 50 years or more old are not uncommon as shade trees in farmyards. As such solitary trees are remote from other sources of pollen it might reasonably be expected that any seeds produced by them would be the result of self-pollination. The primary purpose in seeking such seed was to observe the comparative vigor of selfed seedlings to seedlings of the same species resulting from cross-pollination. In the absence of controlled self-pollinated seed, trees so far removed from other pines as to render negligible the chance of cross-pollination were sought out for seed sources. In the case herein reported the assumption that such trees are necessarily selfed was borne out in an unanticipated manner.

Extraction of the 25 cones produced but 35 sound seeds. This average of 1.4 seeds per cone is possibly indicative of a high degree of self-sterility since it represents something less than a 2 per

cent utilization of cone capacity.

The seeds were planted in early spring of this year and by the end of May germination was completed. Of the 35 seeds planted 25 were observed to germinate and of these 17 seedlings were of normal green coloring while the remaining 8 were albinos. This is a close approximation to the usual 3:1 segregation for albinism, which in this case would have a theoretical expectation of 18.75 normal plants : 6.25 albinos. From these figures it was concluded that the parent tree is heterozygous for a factor for albinism and also that the original assumption of self-pollination was valid.

Another lot of Austrian pine seeds, in this case collected from an individual growing in a group of such trees, produced a number of abnormally pigmented seedlings in a ratio of 112 normal: 13 deficient. The deficient seedlings had normally pigmented cotyledons, but when the primary needles began to develop they were found to be almost without green coloring. At the time of writing (mid-August) six of these abnormal seedlings are living and have developed some pigmentation toward the base of the primary needles. The plants are noticeably weaker than their normal siblings and it is unlikely that it will be possible to keep them alive much longer. Here, due to the position of the seed tree in a group of similar trees, some cross-pollination almost surely occurred obscuring any probable ratio of normal to color deficient plants. It is planned to make some controlled pollinations on this tree at some future time in an effort to determine more exactly the nature of this defect.

Individual albino seedlings were observed this spring in plantings of purchased seed of *Pinus Peuce* and *P. Jeffreyi*. In neither of these cases is it possible to obtain enough information regarding the exact origin of the seed to warrant more than the mere recording of the observation.

A. G. Johnson

Waukesha, Wisconsin

◆

Births at New High in 1947

THE number of births and the provisional birth rate in the Unitel States in 1947 set a new high, the infant mortality rate dropped to a new low, and the death rate for the population as a whole was only slightly above the record low in 1946, according to estimates prepared by the U. S. Public Health Service.

Provisional data indicate that approximately 3,720,000 live births were registered in the United States in 1947, an increase of 431,000 over 1946, the previous high, and 785,000 over 1943, the wartime peak. When unregistered births are included, the 1947 total is estimated at 3,910,000 as compared with a revised estimate of 3,470,000 for 1946.

The 1947 provisional birth rate is estimated at 25.9 per 1,000 population including the armed forces overseas, the highest recorded since the birth registration area was established in 1915 and an increase of 11 percent over the rate of 23.3 in 1946.

The provisional death rate in 1947 was 10.1 per 1,000 population excluding the armed forces overseas as compared with 10.0 in 1946 which was a record low.

While the provisional birth rate was rising to a new high level in 1947, the mortality rate for infants under 1 year of age was dropping to a new low. When adjusted for the changing number of births the provisional rate of 32.6 per 1,000 live births in 1947 was 7 percent lower than the provisional rate (35.1) in 1946. Each month in 1947 was substantially below that for the corresponding month of 1946.

THE LAY OF THE CORN-HUCKSTER

"Whose Bread I Eat, His Song I Sing"

HYBRID corn is an epochal modern scientific development. Corn production during the four years of World War II was over two billion bushels *more* because of hybrid corn—and that on a smaller acreage. This increase was enough to pay the total cost of the Manhattan Project, or, to express the effect more concretely, our meat supply was increased some 30 per cent during the later war years because of hybrid corn. Such an accomplishment is entitled to an authentic, accurate history of how it came about. *The Hybrid-Corn Makers,* by A. Richard Crabb* is not that history.

True, it contains many interesting details as to the development of hybrid corn; some, in fact, that have not heretofore been published. But then, by some direct misstatements of fact, by the gross misplacement of emphasis, and by false implications, it results in a completely distorted picture of the whole. The book is made to seem so authentically historical, that the special interests of the members of the American Genetic Association warrant, or even demand, a somewhat detailed critique of this volume in their JOURNAL.

According to the preface, the idea for the book had its inception in a paper on hybrid corn, presented by the author "several years ago" at a meeting of the Illinois Historical Society. "Much later" the author joined the agricultural department of a Chicago advertising organization. The publisher's book jacket states that this was in 1942, and that the position required the travel necessary to the collection of the source material on which the book is based. In its originating influences, then, the book itself is hybrid—between a learned society and the advertising trade, with trade largely dominant. The hybrid influence, however, remains obvious. Chapters II to VII are a fairly clean-cut history of the earliest origins of hybrid corn. Other chapters are no more than propaganda to "sell" the importance of the larger commercial hybrid seed businesses. Still other chapters are mosaics of facts and fancy in which one can not be sure where reality leaves off and fiction begins. Throughout the book one finds the super-superlatives and double talk characteristic of the advertising trade. To all of these errors of commission, add a tremendous omission, namely, that almost no recognition is given to the contributions of the hundreds of small producers of certified hybrid seed. These producers have cooperated with the state experiment stations since the beginning of hybrid corn. They have been of the utmost importance in bringing hybrid corn into being, and in keeping seed quality up and seed prices down. But they rate no more than mention in *The Hybrid-Corn Makers* of Adnan Crabb.

Chapters II, III, and IV probably will hold most of interest to corn geneticists and breeders. They cover the history of the early inbreeding at the Illinois Station. They show East's connection with that work and tell of his move to Connecticut and of how some of the inbreds were sent to him there by Dr. H. H. Love. In this connection there are interesting side-lights on some personalities and their relation to the course of corn-breeding research. The content of chapters V, VI, and VII needs no special comment. It discusses primarily the work of Shull, Hayes and Jones, although other early inbreeders such as Montgomery and Kiesselbach of Nebraska also are mentioned. Chapters II to VI give a reasonably good picture of the development of hybrid corn up to 1915. Moreover, they give East more

*CRABB, A. RICHARD, *The Hybrid-Corn Makers.* Pp. 331+XXV. $3.00. Rutgers University Press, New Brunswick. 1947.

nearly the credit he deserves than has sometimes been the case. H. K. Hayes also is given at least reasonable recognition for his accomplishments. This reviewer yields to no one in his respect for the work of East and Hayes and its importance to the development of hybrid corn. It seems unfortunate, however, that in the later chapters they are sometimes linked with D. F. Jones in reiterated paeans of praise as the originators of hybrid corn. The caption of "Unexpected Help" for chapter V, covering Shull's work, also seems unfortunate. Shull's paper of 1909 was, after all, the first concrete, specific suggestion for combining isolated inbred lines into first-generation hybrids for practical utilization. Whether East or Shull was first to inbreed corn as discussed in chapter V is beside the point. Inbred lines are not hybrids.

Beginnings

The preface to the book clearly implies that the hybrid-corn makers were sent a preliminary draft of the portions dealing with their work. It is suspected that Hayes did not approve the statement on p. 252, that in Minnesota, "Hayes was unable to make a serious start on corn inbreeding until 1920." Certainly this reviewer saw no draft of the considerable material based on a long conference with him, and which contains gross distortions and misstatements. Some of these are amusing. Others are irritating. Thus on pp. 246-247, Richey is quoted *in extenso* as getting a definite concept and a fixed objective from "Jones' discovery of the double cross and his Mendelian explanation of hybrid vigor." Richey categorically denies the statement as quoted. Jones' suggestion for a mechanical modification of Shull's hybrid corn method did provide Richey with *another* objective; but this was in addition to the objectives he had had in mind when his inbreeding was undertaken in 1916, three years before Jones' suggested modification was published.

Which hybrid-corn makers did, in fact, see a preliminary draft? It would be interesting to know if Jones approved chapter VII, captioned "Yankee from Kansas." There (pp. 88-89), Jones is credited with suggesting the interaction of dominant favorable genes as the explanation for hybrid vigor. The author then notes that this, "was not completely new," but that as suggested earlier by Keeble and Pellew[5] it was too simple. Bruce's contribution of 1910[1,9] is ignored. Ignored too is the fact that Collins[2] showed clearly in 1921 that the earlier explanations of Bruce[1] and of Keeble and Pellew[5] were adequate without the linkage which Jones suggested. As East[3] emphasized in 1936, it was linkage and not dominance that was suggested by Jones.[4] Did Holbert read chapter IX dealing with him and Funk Bros. Seed Co.? On p. 136 in that chapter, it states that Holbert developed inbred Hy. Hy was selected out of the Illinois "High Yield" strain by Dr. A. M. Brunson at the Kansas Experiment Station. This misstatement is repeated (by implication) on p. 137 and on p. 184. Another gross misstatement is made on p. 137. This is that U. S. 13 resulted from an idea of Holbert's and St. John's "for a hybrid that would surpass anything that had yet been developed." There is no doubt that U. S. 13 was and is a great hybrid, but it was conceived and executed by Dr. Merle T. Jenkins, not by Holbert and St. John. Again, we ask, did Holbert approve that statement in advance?

Whether an inbred line was developed in Kansas or Illinois is a simple question of fact. The degree of importance that should be attached to an individual's contribution to a whole program is a question of opinion. The reviewer's considered opinion is that Crabb's book grossly exaggerates the influence of Dr. D. F. Jones in the development of hybrid corn. Thus, it is stated on p. 96 that it "was Dr. Donald Jones who carried by far the greatest load in recruiting the hybrid-corn makers of the corn belt." The hybrid corn breeders of the Corn Belt were primarily the corn breeders for the State agricultural experiment stations and the United States Department of Agriculture. Those who developed

during the 1920's the more important inbreds used in corn hybrids in the early 1930's were: Hayes in Minnesota, Holbert for the U. S. D. A. in Illinois, Hoffer and assistants and successors in Indiana, Jenkins in Iowa, Brunson in Kansas, Brink and Dickson in Wisconsin, and Meyer and Stringfield in Ohio. Add to these, Wallace and Baker in Iowa and Pfister in Illinois, if you wish, and you have the list of those developing inbreds known to be in general use in hybrids in the early 1930's. Jones certainly did not "recruit" Hayes, Holbert, Wallace or Pfister. Furthermore he had no part in promoting the programs in Iowa, Kansas and Ohio; that was done by this reviewer (in cooperation, of course). Brink is quoted by Crabb (p. 201) as saying he got much from a trip to New Haven in 1920, while he was a graduate student at Harvard. Only in Indiana does it appear that Jones had an important influence as a recruiter. There, Hoffer states (as quoted by Crabb, p. 168) that Jones is the father of the hybrid corn.

Jones' contribution to the explanation of hybrid vigor has been discussed. The concept of linkage as a general phenomenon was established by others during the few years following 1911. In 1917 Jones[4] showed how this phenomenon would operate in the segregating generations of a corn hybrid to prevent free recombination and therefore prevent (a) a skew distribution and (b) isolation of a multiple dominant. The failure to get (a) and (b) were the objections raised to the 1910 explanation of Bruce[1] and of Keeble and Pellew.[5] Collins[2] showed that (a) and (b) would not be expected in any event within possible populations, if the number of loci involved was as large, say, as 20.

Double Hybrids

The suggestion for double crossing is in a different category. The use of double crosses undoubtedly advanced the general use of hybrid corn by many years. And it is no matter that double crossing almost certainly would have been suggested by others not too much later. It was Jones who did use it in 1918. But the first commercial hybrid seed sold in the Corn Belt, according to Crabb (p. 152), was a single cross. Moreover, most of the "Hi-Bred" crosses during the early years when they were helping to sell hybrid corn to the farmers of Iowa were three-way crosses. Again, some of the most important sweet corn hybrids are single crosses. Finally, the double cross is only a mechanical modification of Shull's underlying suggestion for crossing inbreds for commercial utilization. To by-pass Shull's contribution and have all hybrid-corn makers no more than disciples of Jones (sometimes of Jones, East and Hayes), as Crabb does throughout his book, is just as much a distortion as it would be to ignore the very important contribution that Jones made. If corn breeding develops into a use of single crosses instead of double crosses, must we then discontinue reference to Jones and name Shull only? We hope not.

The Hybrid-Corn Seedsters

In a general way, a chapter is devoted to each of the so-called "Big Four" hybrid seed companies. The shortest of these, 15 pages, deals with Lester Pfister. Nineteen pages are devoted to "The Story of DeKalb," 27 pages to "Henry Agard Wallace" and his Pioneer Hi-Bred Corn Company, and 37 pages to J. R. Holbert and Funk Brothers Seed Company.

After debunking the widespread public belief in Pfister and in Wallace as the originators of hybrid corn, each is given credit for his actual accomplishments. The sanity of the chapter on Wallace and the Pioneer Company is a welcome relief from the wildly excessive claims for his contributions that so often have been made since he became a public character. Wallace's contributions to the theory and practice of hybrid corn utilization were negligible, but the value of his salesmanship and his constant support for the corn breeding program of the experiment stations and the United States Department of Agriculture was tremendous. In "The Story of DeKalb," use is

made of the ancient advertising technic of "Twenty million Frenchmen can't be wrong." Figures on the rapid increase in DeKalb sales abound. As in the chapter on Pfister, opportunity also is taken to emphasize fully Holbert's service and suggestions to the development of the breeding programs. [An interesting commentary on commercial breeding methods is provided by two items in this chapter: (1, on p. 223), St. John began work for DeKalb in 1937; (2, on p. 226), "By 1939 the hybrids developed by St. John for DeKalb were beginning to come in volume, and production ... climbed again." Certainly DeKalb had hired the fastest "hybrid developer" of which we have record!]

The longest chapter in the book is devoted to Dr. J. R. Holbert and Funk Bros. Seed Co. Here the author hits full advertising stride. One claim of accomplishment follows another. Finally, under a spell of its similarity to *The Hucksters*,[11] the reader finds himself paraphrasing its famous "Love that soap," and reiterating, "Love Dr. Holbert," or "Love Dr. Jim" as the case may be. Some of the claims are well founded. Holbert did make contributions to hybrid corn. Other claims, however, run the gamut through various degrees of exaggeration to some that are absolutely false. They reach a crescendo, perhaps, in the statement that "Holbert was by now [1937] dean of all the active hybrid-corn breeders in the United States" (p. 138). Actually, of course, Holbert was never dean of hybrid-corn breeders, either through length of service or through accomplishment.

Omitting inbred Hy, with which Crabb falsely credits Holbert, there remain the inbreds Ill. A, Ill. R4, Ill. 90, and Ill. L of some continuing usefulness that he did develop. Of these only Ill. A and Ill. R4 can be classed as of any general importance. Contrast that with the product of the Iowa, the Kansas, the Ohio, or the Indiana program, and one finds no basis for deanship. Better still, look to Minnesota where Hayes, the real dean, with his assistants has developed the inbreds needed for a self-contained hybrid industry for that state. Perhaps Crabb means to imply that "deanship" depends rather on scientific contributions. An excellent recent summary on "The Experimental Basis for Hybrid Maize"[10] cites 124 papers. Of these, Holbert is senior author of one and the second or third author of two others. But, as far as one can tell from Crabb's book, research is unimportant: All progress in hybrid-corn breeding methods seems practically to have stopped with the suggestions of Shull and of Jones. On pp. 100-101 is a brief reference to an improvement by early testing. Otherwise there is little or nothing regarding the extensive, and successful, research leading to the improvement of old inbreds and to better ways of finding good new ones.

Of course, information on Funk Brothers is not limited to the one long chapter on Holbert. On pp. 314-316 is a letter from Hawaii and four from foreign countries ordering seed corn from the Funks. These are published to show the spread of interest in hybrid corn beyond our shores! It may be significant, however, that the letters were furnished to Crabb by Funk's sales manager and that the *full name and address* of the firm appears in immediate juxtaposition to the letters.

A Look at "The Record"

Three people have received the major public acclaim as originators of hybrid corn: George H. Shull, Lester Pfister, and Henry A. Wallace. Crabb's book discounts their contributions Shull's by an excessive build-up of Jones' influence, and Pfister's and Wallace's by statements of fact. The stage thereby is set for erecting a new hybrid-corn idol, or "Dean." Pointing out some of the errors in the steps leading to the establishment of this new pantheon has seemed more important than calling attention to minor errors having no apparent relation to that main pattern. The following probably belongs in the minor category. The first hybrid corn breeding in Arkansas was not by L. W. Osborne in 1923 (p. 256). It was done by this reviewer,

beginning in 1916. In 1921, 41 single crosses among 10 of these Arkansas-developed inbreds were tested at Burdette, Ark.[8] Moreover, in 1922 fractional-acre isolated blocks were planted at Burdette for producing a single and a double cross. These plantings were total failures because of various vicissitudes.

Chapter XVII is devoted to selling the importance of the previously existing seed trade in establishing hybrid corn, hence its caption, "New Temple on Old Foundations." It begins with praise for the private enterprise system. It gives credit for the concepts (but not for the breeding) of hybrid corn to the State and Federal stations. It also gives those stations credit for producing foundation stocks (which, of course, connotes State and Federal breeding material). It gives some (but little) credit to individuals and organizations without previous experience in producing farm seed. It then extols the part the commercial seed trade has taken. It seems worth while, therefore, to consider briefly the contributions that have been made to hybrid corn by the seed trade.

In spite of the widely advertised corn breeding programs of some companies, they have produced no hybrids demonstrably superior to those of the State and Federal breeders. And this in spite of the fact that they had (in the Corn Belt) full access to the state and federal inbreds as well as their own. Hence, it seems questionable, to what extent "such men as Gunn, Baker, Holbert or St. John have already demonstrated ability to take over full responsibility for their breeding programs" (p. 287), if that responsibility is to the public.

Not all, but some companies have simply taken State and Federal hybrids and sold them under their private designations. Hybrid U. S. 13 was known to have been entered in the Missouri state tests a few years ago under five different private names. It may also have been entered under others. In this way, a public hybrid is taken out of the free competitive market and transferred to a monopolistic one. For example, certified seed of hybrid U. S. 13 has been bought by the thousands of bushels by companies which never sold any U. S. 13. They did, however, sell a very similar hybrid under a private name, and at a price in excess of the going price for U. S. 13. The value of this "contribution" by the seed trade to the advancement of hybrid corn seems at least questionable.

On p. 265, Crabb essentially gives the seed trade credit for spreading the "benefits" of corn hybrids "all the way from Canada to Texas and from Oregon to Georgia." The 1947 report on corn tests in Georgia[6] is received as this is written. Table I shows the average performance of the three Funk hybrids that were tested in four north Georgia counties. The performance of some adapted southern hybrids and open-pollinated varieties also included is shown for comparison. What were the "benefits" to Georgia farmers from using high-priced seed that yielded less than the better open-pollinated varieties, or less than the State-Federal hybrids?

But these are not exceptional results. Those for Tennessee[7] in 1947 are even more striking. The comparative yields of four hybrids certified as adapted to Tennessee, of two Tennessee certified varieties, and of eight commercial hybrids from the Corn Belt (including U. S. 13) are shown in Table II. The yields are the averages for six locations in the State, with six replications at each place. All entries were available commercially in 1947 and all were bought on the open market, except Dixie 17 and Jellicorse which were obtained from experiment stations. Are the yields of 10 to 20 bushels per acre less than could have been had from adapted corn "the bene-

TABLE I.—The average performance of three Funk hybrids in four North Georgia counties in 1947, with comparable values for adapted Southern hybrids and open-pollinated varieties. From [6]

Entry	Acre yield bu.	Erect plants per cent
Tenn. 15	85.0	72
Dixie 17	84.1	88
Tenn. 10	78.5	84
Jellicorse, O. P.	73.5	84
N. Paymaster, O. P.	70.4	85
Funk 717	64.5	74
Funk 714	65.3	82
Funk 713	62.1	83

fits" that have been spread to Tennessee farmers? Actually, the sale of hybrids like these has done much to delay the acceptance of hybrid corn among the intelligent farmers of the Southeastern States. Moreover, it will not be the seed trade of Mr. Crabb's book that will remedy this. The real benefits of hybrid corn are being spread by the State-Federal workers in this area just as they were previously in the Corn Belt.

The seed trade has performed an important function in meeting the demand for hybrid seed corn as it developed. Most members of that trade have followed the highest ethical standards. If some of the shortcomings are emphasized here, it is because they are omitted completely from Crabb's distorted picture.

A Missed Opportunity

It remains but briefly to analyze the factors implying an authenticity which *The Hybrid-Corn Makers* does not, in fact, possess. The first of these is the reference to the role of the Illinois Historical Society in the book's inception. Much of it was written while the author was in the advertising trade. The second implication of authenticity is the emphasis on the extensive personal interviews, plus the implied opportunity of the hybrid corn-makers to review the material dealing with their work. That opportunity was not universal. Furthermore, even if it had been, the author's lack of first-hand information would not have permitted him to display sound critical judgment in accepting or rejecting statements which might be subject to personal bias. The choice of a university press for publication could be a third factor making for apparent authenticity. To these should be added the characteristic advertising touch of having a testimonial in the "Introduction."

Crabb did well indeed in choosing Prof. H. D. Hughes to write the introduction. Certainly it is extremely doubtful if any one who thoroughly understood the impact of the different forces on hybrid corn development would have afforded the book the unequivocal approval as to accuracy given it by Professor Hughes on pp. XXIV and XXV. His further statement that it "will be more and more appreciated with the passing of the years" unfortunately may prove to be true. It is partly to do what he can to prevent the fulfillment of that prediction that this reviewer undertook the always unpleasant task of being a harsh critic. He has, moreover, attempted to introduce enough of the affirmative as not to be completely destructive. To him, the truly great pity is that with the many facts gathered with so much labor, the author should have missed the opportunity to fill an important need.

The reviewer makes no assertion, express or implied, that the author intentionally distorted the picture in order to give undue credit to the hybrid seed trade or to specific companies or individuals. It is immaterial whether the distortions were "bought like a bullock, hoof and hide" by clients* or whether they were the natural concomitant of first-hand ignorance reacting with the universe in which Crabb moves and has his being. The end result is the same. In his closing argument in a damage suit for the lost lives of sailors sent to sea in a ship foredoomed to sink, Rufus Choate once spoke substantially as fol-

TABLE II.—Average acre yields at six locations in Tennessee of four hybrids and two varieties certified as adapted to Tennessee, and of eight other hybrids sold on the market in Tennessee by Corn Belt hybrid seed producers. From [7]

	Entry	Acre yield bu.
C	Dixie 17	85.9
C	Tenn. 10	84.3
C	Jellicorse, O. P.	75.3
C	N. Paymaster, O. P.	74.3
C	Funk G711	74.1
	Funk G515W	73.8
C	Dixie 44	71.2
	Hunnerkoch H14	67.4
	Funk G94	67.3
	DeKalb 898	66.1
	Pioneer 332	66.0
	U. S. 13	65.3
	Pioneer 303	63.6
	Pfister 170	63.4

C indicates certifiable in Tennessee

*Since this was written, it has been learned that complimentary copies of this book have been sent out by one seed company's associates to workers in agricultural education, extension and research.

lows: She sailed down the harbor, painted and perfidious, a coffin, but no ship. This critique may well close with a paraphrase: It came off the presses, printed and perfidious, a romance, but no history.

Literature Cited

1. Bruce, A. B. *Science.* 32:627-628, 1910.
2. Collins, G. N. *Amer. Nat.* 55:116-133. 1921.
3. East, E. M. *Genetics.* 21:375-397. 1936.
4. Jones, D. F. *Nat. Acad. Sci. Proc.* 3: 310-312. 1917.
5. Keeble, F., and C. Pellew. *Jour. Gen.* 1:47-56. 1910.
6. Lebedeff, G. A., and O. L. Brooks. Ga. Expt. Sta. Circ. 154. 11 pp. 1948.
7. Long, O. H., and S. F. McMurray. Tenn. Expt. Sta. Bul. 206. 20 pp. 1948.
8. Richey, F. D. U. S. Dept. Agr. Bul. 1209. 20 pp. 1924.
9. ———. *Jour. Hered.* 36:243-244. 1945.
10. Sprague, G. F. *Biolog. Reviews.* 21: 101-120. 1946.
11. Wakeman, F. *The Hucksters.* 307 pp. New York. 1946.

Frederick D. Richey

Bureau of Plant Industry, Soils and Agricultural Engineering, U. S. Dept. Agr. and Tennessee Agricultural Experiment Station, Knoxville, Tenn.

✦

A TETRAPLOID SPORT OF McINTOSH APPLES

Haig Dermen and George M. Darrow*

THE McIntosh apple is one of the leading commercial varieties of the United States and one of those most used as a parent in breeding to produce superior varieties. A "giant" sport of McIntosh was discovered by Mr. Roger Kimball, of Massachusetts, and fruit and young trees of it were generously sent to us by Mr. Kimball. Cytological examination revealed that the sport was a diploid-tetraploid periclinal chimera, the epidermis being diploid and the internal tissues of the leaves and the stem tetraploid. Einset and associates, at the New York State Agricultural Experiment Station, Geneva, have found a similar distribution of diploid and tetraploid tissues in chimeral "giant" sports in the Ontario and Wealthy varieties.

Polyploidy has proved to be a factor of great economic importance in major plant crops. A few apple varieties are triploid. Among them the Stayman Winesap and the Baldwin are of great value in commercial apple production. A few triploid apples are known, such as the variety Hibernal, and a species of apple, *Malus coronaria,* but their fruit is of no commercial value and they have little to recommend their use in breeding.

It is hoped that periclinal tetraploids similar to the new McIntosh sport may be discovered in such varieties as Red Delicious, Golden Delicious, Jonathan, Winesap, and others that have proved valuable in breeding on the diploid level. Then, using tetraploid forms of these and the McIntosh sport as parents, we may obtain tetraploid seedlings in quantity and make mass selections on the tetraploid level. At present this McIntosh "giant" sport may be used with diploids to produce triploid seedlings.

*Cytologist and Principal Pomologist, respectively, United States Department of Agriculture, Agricultural Research Administration, Bureau of Plant Industry, Soils, and Agricultural Engineering, Division of Fruit and Vegetable Crops and Diseases, Plant Industry Station, Beltsville, Md.

"THE TRUTH" ABOUT GENETICS

Pravda Discusses "Antipatriotic Acts under the Guise of 'Scientific' Criticism"

On September 2, 1947, the Moscow newspaper Pravda *carried on its front page a long article defining the Soviet position regarding genetics. For its great interest to geneticists, and for its news value to all scientists, we are publishing a translation in full.* ED.

CERTAIN English and American journals with pretentious scientific names have been carrying in recent years a wrathful, slanderous, and completely unscientific propaganda against progressive Soviet biological science. The February issue of the English journal *Discovery* contains an article, "Revolution in Soviet science" by a scientific reactionary, C. D. Darlington. This member of the Royal Society has seen fit, by a single stroke of the pen, to liquidate Russian biological science, just as the fascist politicians have in their time "abolished," on paper, the Soviet Union, transforming it into a . . . "geographic concept." Darlington has maliciously declared that Timiriazev's discoveries "are nothing but the requirements of William of Occam," of a medieval scholastic. Darlington calls Michurin an importer of plants" from Canada and U.S.A.! He attacks T. D. Lysenko in the same style.

The slanderous article of Darlington has provoked a justified resentment among English scientists. Angry letters with sharp protests against this article, against the despicable slanderous attacks on Soviet scientists, poured into the editorial office of *Discovery*. H. G. Creighton wrote about the article of Darlington: "the political irritation and the vindictive anticommunist and antisoviet wrath have blinded his objectivity and have led him to some very unscientific arguments against Lysenko." Another American scientist, M. Hamilton, declared to the editor that, "a disagreement in the field of genetics can not serve as a justification for dirty political insinuations," and that Darlington has pleased by his articles only those who "in the depth of their soul hate science and all that it has given and gives to mankind."

One can add nothing to these truthful and objective evaluations of Darlington's article. They clearly show the inner motivation of the attacks on Lysenko and other Soviet biological scientists.

Darlington is seconded in U.S.A. by a certain Professor Karl Sax, who is angered by a mere mention of dialectic materialism and its application in the field of genetics. He is displeased especially because Soviet scientists consider fideism, i.e., the "doctrine" which replaces knowledge by faith, to be foolishness. He endorses the racists demagogic assertions well known to the Soviet people, that our country is "totalitarian" and that biological science in it is "suppressed."

Karl Sax's effort appears in the American journal *Science*. It is notable that in U.S.A., as well as in England, a series of scientists have protested against Sax's reactionary political insinuations. Dunn, a professor of Columbia University, in his reply to K. Sax in the same journal declared: "The progress of the biological research in the Soviet Union gives us a very valuable lesson. Great individuals have arisen in Soviet biology, valuable discoveries have been made and continue to be made now in the midst of war" (*Science*, 1944, No. 2561, p. 67).

J. Somerville, an American scientist who attended the discussion of the problems of genetics in USSR in 1939, wrote: "So long as representatives of the state act according to the principles of dialectic materialism, there can be no question of scientific conclusions made

under dictation, or of an artificial limitation of scientific activity. The dialectic materialism is a philosophic doctrine which not only does not hold science in contempt but considers it the basic source of its own principles" (*Philosophy of Science,* 1945, Vol. 12, No. 1).

In the light of all this, a feeling of deep indignation is provoked by the debut of A. Zhebrak, a member of the White-Russian Academy of Sciences, on the pages of the journal *Science* with an anti-patriotic article, "Soviet Biology," directed against progressive Soviet biological science.

One could expect that the Soviet scientist, A. Zhebrak, would stand against the dirty slander on Soviet science made by reactionary English and American "scientists," such attacks on Soviet science as, according to the justified remark of the English biologist Haldane, came in the past "from Hitler's friends." Unfortunately, we see the exact opposite. Under the guise of polemics with Prof. Sax, A. Zhebrak has completely adopted the latter's stand. Together with the most reactionary foreign scientists, he humiliates and defames our progressive Soviet biological science and its eminent modern representative, academician T. D. Lysenko.

First of all, A. Zebrak has completely misrepresented Soviet biological science before the American reader. In dealing with problems of plant physiology, genetics, and selection, he has deliberately omitted to mention such leaders of science as Timiriazev, Michurin, and Williams, whose work is the basis of that of the modern Soviet investigators in this field. Not a word is said about such world-renowned scientific institutions as Lenin's All-Soviet Academy of Agricultural Sciences, the Institute of Genetics of the Academy of Sciences of USSR, and others.

Solidarity with the reactionary K. Sax is undignified for a Soviet scientist, A. Zhebrak. Writes A. Zhebrak: "Unfortunately, Professor Sax . . . regards modern biology, and particularly genetics, as synonymous with the name of the academician Lysenko. In criticizing the mistaken views of Lysenko, he apparently holds the opinion that he criticizes not only the academician Lysenko but also the very basis of Soviet biology and the relation of the Soviet government to science."

One must have lost the feeling of patriotism and of scientific honesty to declare that a scientist known to the whole world, a pioneer in the field of genetics, academician Lysenko, has no relation to Soviet biology. . . . What a feeling of patriotic indignation will be the answer to this of every Soviet peasant, every writer in socialist agriculture, who for a number of years has been getting good harvests on the basis of scientific methods developed by T. D. Lysenko!

A. Zhebrak has needed this in order that, having given over the Soviet scientist T. D. Lysenko for the amusement of the corrupt capitalist press, he might represent himself a devoted ally of Darlingtons, Saxes and their like, who chose to "criticize" T. D. Lysenko in order to fight for reactionary idealism against dialectic materialism.

The political meaning of this fight is obvious to everyone. Reactionary biologists know it well. None other than Robert Simpson, in an article directed against Lysenko and entitled "Science—according to the totalitarian model," has declared, "The dual role of Lysenko in politics and science calls for a fearless inquiry by scientific men into the dangers of science becoming subservient to the state. . . . It's time to break a few lances." Simpson is worried because the Soviet land has found methods to get an abundance of grain, milk, meat, wool, and fur. He is worried because capitalism is unable to give this abundance to the people.

Zhebrak, as a Soviet scientist, ought to have unmasked the class meaning of the struggle taking place in connection with problems of genetics. But, blinded by bourgeois prejudices, by a contemptible subservience to bourgeois science, he has adopted the view of the enemy camp. To please this camp, he has dis-

honored representatives of Russian science in a foreign journal which specializes in maligning Soviet scientists. For A. Zhebrak there exists such a thing as "pure science." Zhebrak writes: "together with American scientists, we, working in the same field of science in Russia, are building a general biology on a world-wide scale."

So, this is the source of the antipatriotism of this "scientist"! So, neither does progressive Soviet biological science exist in nature, nor is there a reactionary idealistic biology. So, there exists only a single biology "on a world-wide scale." This explains the alliance of Zhebrak with Darlington, Sax, and other obscurantists of the reactionary capitalistic camp.

A national exclusiveness is remote from Soviet scientists. In their investigations, they generalize everything progressive which is given by the foremost scientists of foreign lands. They develop science on the basis of comradely criticism and self-criticism. They are imbued with the spirit of life-giving Soviet patriotism, the spirit of Soviet national pride in their science, science which develops on the only solid foundation, dialectic materialism,—science which serves the interests of the people. A Soviet scientist is proud to wage an unrelenting struggle with that "science" which is profoundly inimical to dialectic materialism, which serves not the people but the fooling of people, and the strengthening of the power of exploiters.

The antipatriotic act of A. Zhebrak is aggravated by a personal attack on the academician T. D. Lysenko. With a petit-bourgeois impertinence, he declares on the pages of an American journal that academician Lysenko was rewarded by the government "not for his opinions or experiments in the field of genetics" but only "for his work in the field of agricultural practices." Knowingly misrepresenting the orders of the Presidium of the Supreme Soviet of USSR regarding the bestowal of high government rewards on the academician Lysenko for eminent scientific attainments, for theoretical scientific works, which have a first rate importance in the practice of socialist construction, A. Zhebrak is calumniating Soviet science by this dishonest declaration. More than that, he makes the absurd assertion that the work of academician Lysenko ". . . founded in reality on naive and purely speculative conclusions, is unable to interfere with successful development of genetics in USSR." It follows that, according to Zhebrak, Lysenko acts as a brake on progressive development of genetics! A. Zhebrak has gone too far.

How disgusting is the role of Zhebrak in taking it upon himself to please the reactionaries of the whole world by defamation of his compatriot scientist in the pages of a foreign journal inimical to us! Only a man who has lost all sense of social duty could take such a step.

One could close the discussion of this antipatriotic attack by Zhebrak, but we have before us another issue of the American journal *Science,* Vol. 105, No. 2718, for 1947. It has an article by N. P. Dubinin (Institute of Experimental Biology of the Academy of Sciences of USSR), entitled "The work of Soviet biologists: theoretical genetics."

First of all, one is put on one's guard by the fact that this journal, inimically disposed toward Soviet biologists, gives a front place to Dubinin's article. But the answer comes easily. All the founders of Soviet biology are ignored in the article. Whom does Dubinin regard as Soviet biologists? It happens that here are lauded such "Soviet biologists" as Dobzhansky, Timofeef-Ressovsky—open enemies of the Soviet people.

Dobzhansky, for example, is now working in U.S.A. in the field of slander against Soviet biologists and "creates the style" in this anti-Soviet campaign. With a feeling of loathing and indignation one reads further in this article praises for a series of similar personages. The whole Michurinian trend in biology is ignored by Dubinin. Every Soviet biologist will resolutely rise against such an obviously wrong, clear-

ly antipatriotic, representation of Soviet biology.

What do the above facts mean? They mean that a certain backward part of our Soviet intelligentsia still carries a slavish servility for bourgeois science, which is profoundly foreign to Soviet patriotism. We are proud of our country, of victorious socialism, and of our progressive Soviet science. It is necessary to pull out decisively and pitilessly the rotten roots of obsequiousness and slavishness toward bourgeois culture. Only in such a way can one accomplish successfully the great task set before Soviet science by Comrade Stalin, namely, to exceed in the briefest possible time the attainments of science in foreign lands.

To the court of public opinion with those who act as a brake on the accomplishment of this task, those who defame our progressive Soviet science by their antipatriotic acts!

PROFESSOR I. LAPTEV

Pravda
September 2, 1947

New Soviet Ouster Caused by Article

UNDER the above headline the *New York Times* of February 10 reported the dismissal of Anton R. Zhebrak from his post as president of the White Russian Academy of Science. His downfall was interpreted as a direct consequence of the defections from orthodox Soviet biology charged against him in Prof. Laptev's Pravda blast. The Paris dispatch which disclosed the news of Zhebrak's ouster said that he had been replaced by Nikolai Ivanovitch Grashchenkov, identified as "a former party propaganda expert." The *Times* article, somewhat condensed, is reproduced below.

Professor Zhebrak made the mistake of contributing an article to the Oct. 5, 1945, issue of Science, the official organ of the American Association for the Advancement of Science, in which he defended Soviet science but at the same time spoke slightingly of T. D. Lysenko, the all-powerful director of the Institute of Genetics of the Soviet Academy of Science. This brought down on his head a violent denunciation by Prof. I. Laptev in *Pravda* [which is printed above in full].

The same article also sharply criticized another famous Soviet biologist, N. P. Dubinin, for having contributed an article to Science last year on the work of Soviet biologists.

This newest episode is a continuation of a scientific battle that has been raging for nearly fifteen years and has attracted world-wide attention. It began with the presentation by Lysenko in 1936 of a theory that classical genetics—the genetics of Mendel and Morgan—was false because it failed to conform to dialetical materialism.

A heated controversy ensued in which leading Russian geneticists took issue with Lysenko's theories. The best-known Soviet geneticist of the time was Nikolai Vavilov, president of the Lenin Academy of Agricultural Sciences and director of the Institute of Applied Botany. He was so highly regarded by geneticists in other countries that he was elected president of the International Genetics Congress in Edinburgh in 1939, although he was not permitted to attend it.

Lysenko and his followers were eventually victorious, partly on the ground that Mendel was a priest. Lysenko supplanted Vavilov as director of the Genetics Institute of the Academy of Science and was hailed widely in the Soviet press as the discoverer of "vernalization," which his followers claim increased crop yields, but of which scientists elsewhere have been skeptical.

Lysenko went on to new honors, being named vice chairman of the Supreme Soviet and receiving the Order of Lenin, while the unfortunate Vavilov lost his posts. In 1940 Vavilov was arrested and spent the winter of 1941-42 as a prisoner in a concentration camp at Saratov, according to an article in last August's *Journal of Heredity*, by Prof. Theodosius Dobshansky of Columbia University. Vavilov died in Siberia n 1942, according to this article, which said that he "suffered martrydom for genetics."

The disputes engendered by Vavilov's death was continued in various scientific publications. Dr. Karl Sax, Professor of Botany at Har-

vard, contributed an article to "Science" in 1944, which charged that "science must conform to political philsopohy" in the Soviet Union.

In reply to this article, Professor Zhebrak contributed the article that led to his downfall. In it he contended that "science can be free in a centralized socialistic state." He made the blunder, however, of adding:

"Unfortunately, Professor Sax * * * regards modern biology, and particularly genetics, as anonymous with the name of the academician Lysenko. In crticizing the mistaken views of Lysenko, he apparently holds the opinion that he criticizes not only the academician Lysenko, but also the very basis of Soviet biology and the relation of the Soviet Government to science."

This quotation was cited in Pravda as showing Professor Zhebrak's "solidarity with the reactionary K. Sax."

Unamericana

THE use of the public press to defame character, to impugn loyalty, to impute heresy, is not restricted to Russia alone. Denunciation by headlines is a device of demagogues everywhere. The recent charges levelled against Dr. E. U. Condon by the Committee on Un-American Activities provide us with an unhappy instance here at home. Scientists everywhere, singly and in groups, have raised their voices in protest against the pillorying of Dr. Condon. What is at question is not the right of the Government or the Congress to safeguard the national security, but whether an individual may be denied the right to defend himself against accusations made against him. The following resolution is one action of many taken by American scientists against the Committee's summary action.—ED.

The American Society for Pharmacology and Experimental Therapeutics, Inc., hereby records its strong disapproval of the methods employed by the House of Representatives Committee on Un-American Activities in publicly implying that Doctor E. U. Condon, Director of the National Bureau of Standards, had failed properly to safeguard secrets vital to the safety of the United States. The Committee on Un-American Activities violated basic civil rights in thus making implied accusations not proved by facts in evidence. The Society wishes to emphasize that the fabric of cooperation between scientists and their government, so carefully and strongly woven during the war and so important for the welfare and safetly of the United States, will be seriously weakened if the loyalty and integrity of citizens can be questioned without furnishing acceptable substantiating evidence in conformity with established judicial procedures.

Atlantic City, March 18, 1948.

The Executive Board of the Washington Association of Scientists has prepared an eight page mimeographed analysis of the sequence of events and their implications. It concludes with this succinct warning:

Finally, there needs to be mentioned a general implication of the attack upon Dr. Condon which goes beyond the problems of science and scientists. On the basis of evidence which the Subcommittee admits is insufficient to sustain a charge of disloyalty, it nevertheless recommends that the Secretary of Commerce dismiss Condon or publicly defend his failure to do so. This obviously constitutes an attempt, through the use of unfounded charges and innuendo, to so inflame public opinion that Condon will be separated from Federal service. This impatience with orderly legal procedure and resort to direct action is, in other contexts, the pattern of vigilanteeism and similar lawlessness. When it occurs under the sanctions of governmental authority it is not easily distinguishable from the very types of Un-Americanism which the Committee was established to combat.

The implications of the attack upon Dr. Condon by the House Subcommittee on Un-Americanism Activities are therefore dangerous and ugly. Seen as one element in a current broad trend, they threaten not only one scientist and public servant but the security and fundamental tenets of a democratic people.

FURTHER DATA ON SYMPHALANGISM

ARTHUR G. STEINBERG and EARLE L. REYNOLDS*

SYMPHALANGISM involves the fusion, usually congenital, of the middle phalanx with either the proximal or distal phalanges in one or more fingers or toes. The pedigree we are describing was the subject of a brief note in this JOURNAL by Inman[6] in 1924. In consequence, this report is to some degree an extension of the earlier report. The pedigree as far as it could be traced is shown in Figure 2.

The proband in the earlier report was at that time a student at the college. He showed then (and now) fusion of the middle and distal phalanges of the second and fifth fingers of both hands. The second, third, fourth and fifth toes of both feet were also affected. (See Figures 8 through 11 in Inman's report.) The erroneous impression is gained from the earlier report that in this pedigree the effect on the hand is limited to the second and fifth fingers (for example, see Gates[4] p. 400). That this is definitely not so can be seen by reference to Figure 3 in the present paper, which presents x-rays of the hands of the mother (II-4) of the original proband. There is evidence of a distal joint only in the fourth digit of the left hand. This joint can be flexed slightly; and the skin over this joint, unlike that over the distal joints on the other fingers, shows some of the wrinkling which is characteristic of normal joints (See Figure 4).

The mother's hands show another anomaly not exhibited by those of her son or affected grandsons, namely, a short index finger. This shortness is due to the great reduction of the distal phalanx of the index finger (Figure 3). Her brother (II-3), now dead, is reported to have had a short index finger also. This condition has been reported in other pedigrees of symphalangism (See Gates[4] for review). Unfortunately, the details of how other members of the family were afflicted are not available.

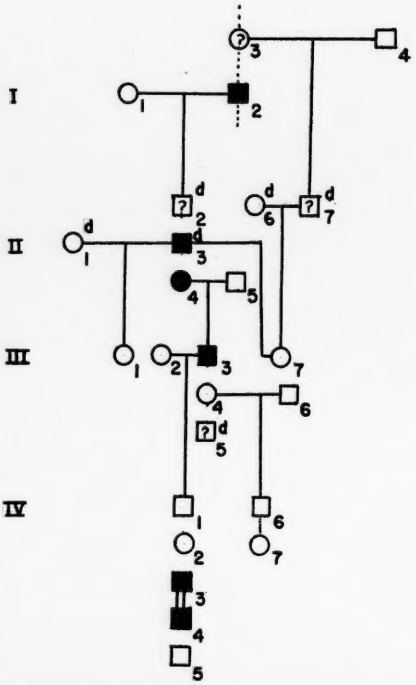
Case I-2 is known to have had six sisters and three brothers. They are all dead and information concerning them or their children is not available. It is likely, however, that I-3 had normal hands, since III-7, who is still living, was unaware of the "family trait" until she married II-3. Case II-2 died at three years of age and nothing is known about his hands.

The twin boys, IV-3 and IV-4, were brought to the Institute for diagnosis as to their zygosity. In the course of this examination, stiffness of the distal joints in the second and fifth digits of both hands was observed in one of the twins, Tim. The other twin, Ted, can flex the terminal joint of the fifth digit on his right hand. X-rays of the children's hands (Figures 5*A* and *B*) show no evidence of fusion of the distal and middle phalanges. It is likely that such fusion will occur at a later age; they were only three years old at the time.

The x-rays of the twins' hands show two other anomalies: an extra epiphysis at the proximal end of each second metacarpal, and an extra epiphysis at the distal end of the middle phalanx of the second finger of each hand. The x-rays of the hands of 167 other children from the Fels series were examined for these anomalies. The first appeared in only six of the 167 children (3.6 per cent), while the second did not appear in any of the x-rays examined. While it is possible that the extra epiphysis at the distal end of the middle phalanx of the second finger is part of the syndrome of symphalangism, it is highly unlikely that the other extra epiphysis is part of the same syndrome, because of its relatively frequent occurrence in the absence of symphalangism.

*From the Fels Research Institute for the Study of Human Development, Antioch College, Yellow Springs, Ohio.

Symphalangism has been reported frequently in the past under a variety of names, e.g., anarthrosis, synostosis, hereditary ankylosis, and several others. None of these reports, except that of Inman (cited above) concerns a case which involves fusion of the middle and distal phalanges of the second and fifth fingers only. There are several reports concerning fusion of the middle and distal phalanges of *either* the second or the fifth fingers. For extensive reviews of the literature see Daniel,[2] and Freud and Slobody.[3] In every case the character is due to a dominant mutation. The expression of the character as reported in the different published pedigrees is sufficiently variable within a pedigree (except in short pedigrees involving few individuals) to make it difficult to hazard a guess as to how many different gene loci are concerned in these several pedigrees.



PEDIGREE OF SYMPHALANGISM

Figure 2

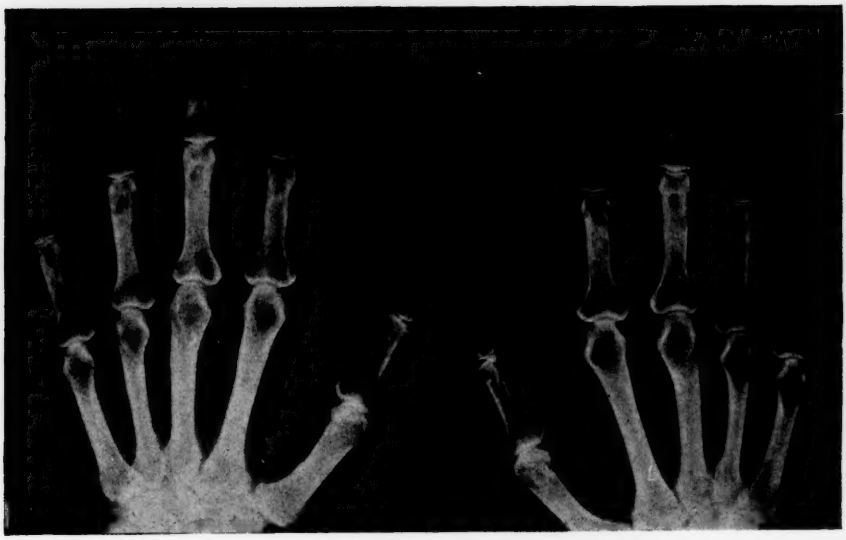
In this pedigree chart the children of a union are arranged vertically with the eldest child at the head of the column. See text for further details.

Several Genes for Symphalangism

In other pedigrees presented in the literature, different digits may be affected in different individuals, but the affect always involves either the middle and proximal phalanges or the middle and distal phalanges. Hence it seems highly improbable that all genetically determined cases of anarthrosis are the result of mutation at the same locus. Those cases involving fusions of the middle and distal phalanges are most probably caused by a mutation—or mutations—at a locus different from those involving fusions of the proximal and middle phalanges. No case is reported in which one member of a pedigree has the proximal joints affected and another the distal joints.

The case reported by Freud and Slobody[3] (see the x-rays of the four children in one family of their pedigree) shows such an unusual phenotype as to require classification distinct from either of these categories. We would expect it to be due to a mutation at a different locus from the other two already postulated. It should be recognized that three loci are a minimum estimate of the number of loci concerned, for identical phenotypes frequently may result from the action of distinct genotypes.

Correspondence with R. A. Hefner indicates that it is unlikely that this family, the initial of whose last name is B, is related to that studied by him[5] and by Cushing.[1] The family names are different, and the present family migrated to Michigan from Birmingham, England, about 1841. The family reported in the earlier papers traces back to Scotland. In addition, the middle and proximal phalanges are affected in the earlier pedigrees, whereas the middle and distal are affected in the present one. As stated above, this strongly suggests that different genes are concerned in the two pedigrees.



X-RAYS OF THE HANDS OF II-4

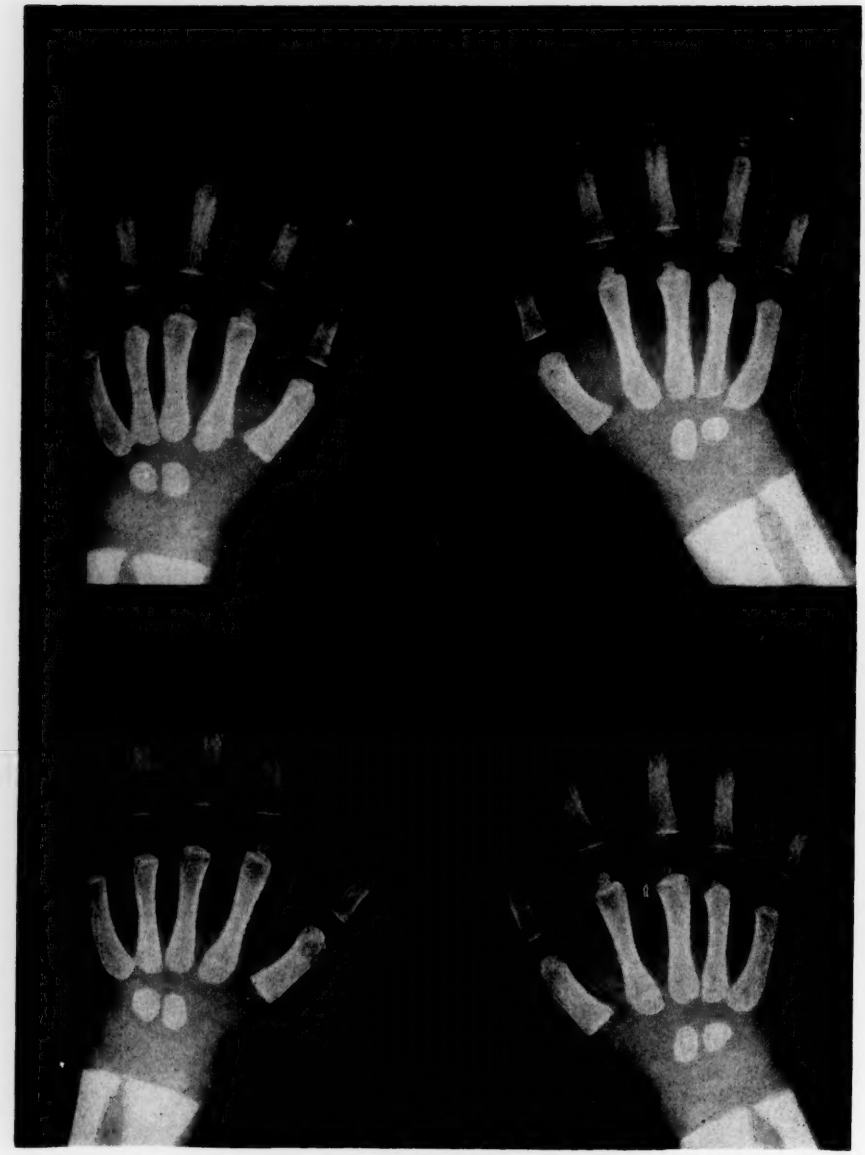
Figure 3

Note complete fusion of middle and distal phalanges of all digits except the fourth of the left hand. The distal phalanx of each index finger is abnormally short.

PHOTOGRAPH OF THE HANDS OF II-4

Figure 4

Note short index fingers and the presence of wrinkling at the distal joint of fourth digit of left hand, and its absence at all other distal joints.



THE HANDS OF THE B TWINS

Figure 5

X-rays of hands of Tim and Ted, respectively. Note similarity of bone formation, including an extra epiphysis at the proximal end of the second metacarpal bone in each hand, and an extra epiphysis at the distal end of middle phalanx of second finger of each hand. The middle phalanges of "unaffected" fingers are abnormally short.

Time of Joint Fusion

The present pedigree sheds light on the question raised in the past, as to whether the immobility of the joints observed in symphalangism preceeds or is the result of the fusion of the phalanges. It is evident from the x-rays of the twins' hands that immobility, at least in some cases, precedes fusion of the phalanges. The affected joints in the fingers of the twins are rigid and cannot be moved passively, that is, rigidity is complete. Nothing is known of the morphological features underlying this condition. While it can be stated that rigidity precedes bony fusion, it cannot be said that the fusion is caused by the rigidity. For example, we have one case in our files of an adult with congenital rigidity of the ring finger of each hand, in the absence of bony fusion.

It should be noted in passing that the middle phalanges of the unaffected fingers of the twins (Figure 5 *A* and *B*), and of their father (Figures 8 and 9 in Inman) are considerably shorter than those of normal hands. Daniel[2] reported the same condition in his pedigree and presents measurements of the hands of normals and affected individuals to establish the point. Because of the small size of our sample, and because of the obviousness of the difference, we did not believe it worth while to conduct a detailed investigation of this point.

Freud and Slobody[3] found in their pedigree of symphalangism that the fusion of the proximal and middle phalanges became progressively less marked as one progressed from the fifth toward the second finger of a given individual. Fusion was complete in the former and is evidenced by only a slight bony bridge in the latter. This was true of the hands of all four children whose x-rays were published, including that of an 18-months old girl. On the basis of the above observations these authors state:

> It should be emphasized that symphalangism is due to failure of differentiation of the interphalangeal joint during the embryonic stage. The previously held theory, that ossification of existing joints takes place in the fetus, is untenable. The roentgenograms demonstrate the stages of articular differentiation. The earliest stage is a constriction of the phalangeal beam. This progresses until only a central bony bridge remains. This bridging becomes less distinct and then invisible, and then a normal joint space is formed. The process may be stopped at any point before complete differentiation, with resulting symphalangism.

It seems to the present authors that Freud and Slobody's viewpoint does not follow from their data, since if the x-rays are read from the second finger toward the fifth finger a conclusion exactly opposite to theirs, and in agreement with well established embryological observations, may be drawn. Finally, the condition of the hands of the twins in our study indicates that symphalangism is not due "to the failure of differentiation of the interphalangeal joint during the embryonic stage," but rather to rigidity followed by fusion of the originally independent phalanges.

Summary

1. Symphalangism is traced through another generation of an earlier pedigree.

2. Evidence is offered that rigidity of the joint precedes fusion of the phalanges.

3. It is estimated that mutations at any of at least three different loci can cause symphalangism.

Literature Cited

1. Cushing, Harvey. *Genetics* 1:90-106. 1916.
2. Daniel, G. H. *Ann. of Eugen.* 7:281-297. 1936.
3. Freud, Paul and Lawrence B. Slobody. *Am. J. Dis. Child* 65:550-557. 1943.
4. Gates, R. R. Human Genetics. 2 vols., Macmillan Co., N. Y. Pp. 1518. 1946.
5. Hefner, R. A. *J. Hered.* 15:323-329. 1924.
6. Inman, O. L. *J. Hered.* 15:329-334. 1924.

LINKAGE OF WHITE AND WAVED—1

HELEN BUNKER AND G. D. SNELL
Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine

WHITE is a new dominant mutation in the mouse recently described by Grobman and Charles*. Homozygous animals appear like albinos, having white coats and pink eyes. The heterozygotes are craracterized by a general lightening of the coat color, usually accompanied by a white belly spot of varying size.

To test for linkage of white with other known genes, mice with the white mutation (*Whwh*) were crossed to animals of the following stocks:

CA	*CaCa Fufu Ww*
P	*aa bb dse/dse pp rr*
RE	*ReRe Spsp*
V	*aa lnln ss wa-1 wa-1 vv*
WA	*ff sh-2wa-2 sh-2wa-2*

The recessives belted (*bt*), albino (*c*), and fuzzy (*fz*) were also tested for linkage with white. Backcross data exclusively were used in computing the number of crossovers. The results are summarized in Table I.

Summary

White is linked with waved-1, the total crossover per cent being 8.98%. Although the data show the reverse of the usual sex difference, this variation is not statistically significant. When the female was the heterozygote in the backcross the crossover per cent was 8.7%. Heterozygous males gave 10.0% crossing over.†

TABLE I.—Linkage Data and Crossover Percentages

Gene	Mating*	++	χ+	+*Wh*	χ*Wh*	Total	C-O.-%
b	C	43	41	51	47	182	49.5 ± 3.7
bt	C	18	17	17	15	67	49.3 ± 6.1
c	C	50		35		85	58.8 ± 5.4
Ca	R	21	36	17	24	98	45.9 ± 5.1
Ca	C	4	3	6	4	17	52.9 ± 12.1
dse	C	21	29	33	22	105	41.0 ± 4.9
f	C	43	34	49	29	155	46.5 ± 4.0
Fu	R	17	12	16	2	47	40.4 ± 7.3
fz	C	29	21	20	15	85	51.8 ± 5.4
ln	C	67	69	59	65	260	50.8 ± 3.1
P	C	26	24	27	28	105	51.4 ± 4.9
r†	R	34	16	38	15	103	47.6 ± 4.9
Re	R	46	41	45	43	175	50.9 ± 3.8
s	C	71	65	54	70	260	54.2 ± 3.1
sh-2	C	41	34	51	29	155	45.2 ± 4.0
Sp	R	43	44	54	34	175	44.0 ± 3.8
v	C	71	66	70	53	260	47.7 ± 3.1
W	R	23	34	21	20	98	43.9 ± 5.1
wa-1	C	5	94	85	12	196	8.7 ± 3.6
(♀ *wa-1*+/+*Wh*)							
	C	6	31	27	0	64	10.0 ± 6.2
(♂ *wa-1*+/+*Wh*)							
wa-2	C	47	28	54	26	155	47.1 ± 4.0

*All matings are backcrosses. C = coupling; R = repulsion phase.

†The ratio *R*:*r*::72:31 suggests that some or all of the P stock mice used for the backcrosses were *Rr* instead of *rr*. Unfortunately they were killed before this could be tested.

*ARNOLD GROBMAN and D. R. CHARLES. *Jour. Hered* 38:381-4. 1947.

†This investigation has been aided by a grant to the Roscoe B. Jackson Memorial Laboratory from the National Cancer Institute.

✦

Dental Caries, Nutrition and Heredity

THE effects of nutrition and heredity on the incidence of dental caries are discussed in the February issue of *Current Research in the Science of Nutrition*. A survey of 3,900 persons made by the Italian Medical Nutrition Mission in 1945 revealed a singularly low incidence of caries among the Italians as compared with similar age groups in the United States. The fact that the people studied were regarded by the investigators as "relatively malnourished (perhaps in regard to calories chiefly) and oral hygiene was not commonly practiced" might lead to the conclusion that there is a genetic component for resistance to dental caries.

At first glance this seems to be borne out by the findings of G. W. Clapp who recorded a low incidence of tooth decay in young adult Italians born in Italy and living in the United States. However it is noted that these individuals had maintained Italian dietary habits, suggesting that the low incidence might be correlated with nutrition.

A third group consisting of 500 children of Italian descent was studied by C. D. M. Day and H. J. Sedwick. These children "whose diets apparently had been Americanized from infancy" showed "an incidence of carious lesions which was entirely comparable to the caries incidence of American children.

It is concluded that "these data indicate that the dietary habits are of greater significance than the hereditary influence, although the latter may be significant when the dietary habits are borderline in regard to caries production."

INHERITED VISCERAL INVERSION IN MICE

JOSEPH A. TIHEN,*
DONALD R. CHARLES
AND
THEODORE O. SIPPEL

Manhattan and Zoological Departments, University of Richester

SITUS INVERSUS VISCERUM is rare in mice, as in men. No case was found among more than 11,500 *dba* × C57 mice autopsied in this laboratory.† Human frequencies ranging from 3×10^{-5} to 7×10^{-4} have been reported (Cockayne 1938).

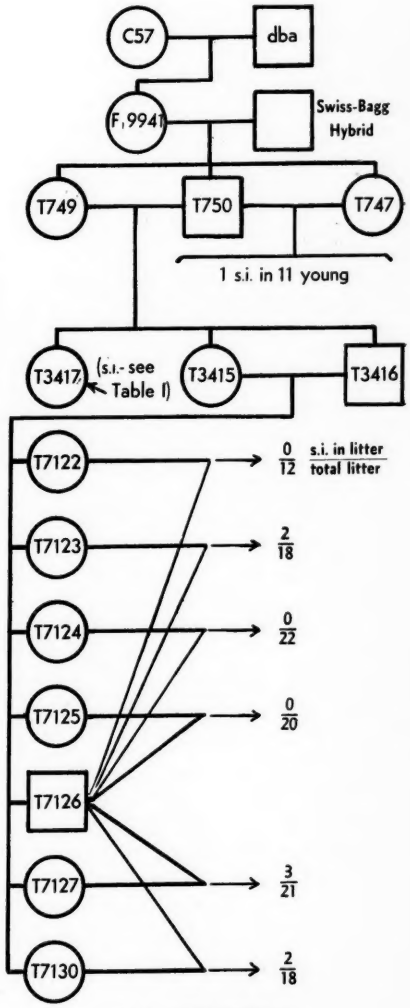
In man, the anomaly is determined by a single autosomal recessive gene.[1] That it might have a similar basis in mice was suggested by an observation of Dr. Katrina Hummel,‡ of the Jackson Laboratory. She found seven cases among 250 Bagg albinos, but none in a number of other strains. The Jackson Bagg line may, in fact, have been the source of the situs inversus gene to be reported here.

The anomaly first appeared in our laboratory as indicated by the pedigree (Figure 6).

The line has been carried three generations further with situs inversus behaving as though determined by an autosomal recessive gene, as in man. For the mutant we shall use the symbol *v*.

Among 24 tested normal offspring of $+/v \times +/v$ there were 9 $+/+$ and 15 $+/v$. Among 19 tested normal offspring of $+/+ \times +/v$ there were 9 $+/+$ and 10 $+/v$.

Among 227 offspring of $+/v \times +/v$, situs inversus has been detected in only 29 or about half of the expected number.



S.I.V. PEDIGREE

Figure 6

Originating from a cross of two Jackson Laboratory strains, 29 cases of visceral inversion have been observed.

*Now of the Department of Zoology, Tulane University.

†Results here reported were obtained incidentally to work carried on for the Manhattan Project at the University of Rochester under contract number W-7401-eng-49.

‡Personal communication.

Causes of the deficiency have yet to be studied, but two seem probable: (1) pre-natal mortality, since the observed *vv* animals were small and weak at birth and died within a few weeks; (2) some variation of the *vv* phenotype, so that not all *vv* animals are detected by external examination shortly after birth.

Salient features of six *vv* and two probably *vv* animals are shown in Table I. Throughout there is a striking similarity to human cases of situs inversus: in main features,[4] in variability of expression, and in association with other, not obviously related anomalies — here hydrocephalus chiefly, in man a wide variety of anomalies including hydrocephalus. This association may in part account for the lowered viability of *vv* in men.[1]

Conclusion

Action of the *v* gene adds a fourth way of producing situs inversus in experimental animals: the other three are by natural or induced twinning, by reversal of archenteron roof and by intro-

TABLE I. Detailed description of eight individuals showing visceral inversion.

Phenotypic expression	situs inversus viscerum perfectus						situs inversus viscerum partialis	
	♀ T11035	T11401a	T11401b	♂ T12321	♂ T12287	♂ T13493	♀ T3417	♀ T14462
general habitus: small	+	+	+	+	+	+	+	+
sickly-looking	+	+	+	+	+	+	+	−
cranium swollen	+	+	+	+	+	+	−	+
skull cap thin, distended	+	+ (foramen between parietal and interparietal)	+	+	+	+	− (very thick)	+
brain soft and hydrocephalic?	+	+	+	+	+	?	+	?
heart: chambers and position reversed aortic arch and branches reversed	+	+	+	+	+	+	−	−
lung lobes	2L,1R	3L, 1R	3L,1R,2C	2L,1R,2C	3L, 1R,2C	3L,1R	1L,1R	normal
stomach reversed	+	+	+	+	+	+	−	−
spleen	in two parts; one to L, one to R of stomach	as in 11401, but more dorsal	dextral, in mirror image of normal relation to stomach	dorsal to stomach; embedded in pancreas	dorsal to stomach	like 11401b	embedded in pancreas; dorsal to stomach	small; in normal position
kidneys: R anterior to L	+	+	+	− at same anterior-posterior level	+	+	−	− (like 12321)
adrenals reversed	+	+	+ (L adrenal lateral to V. postcava)	−	+	?	−	+
veins of renal region reversed	+	+	+	L & R renal veins enter V. postcava at same level; others bear normal relation to V. renal of same side	+	+	−	−
postcaval vein continuous with	V. hemiazygos	V. hemiazygos	?	V. azygos	?	?	V. azygos	V. hemiazygos
other features	no V. hemizygos	—		no V. azygos	?		no V. hemiazygos	portal vein passes ventral to duodenum

duction of unusual optical isomers of protoplasmic components (literature references in Wood and Blalock,[4] Gray,[2] Lynn.[3]) Whether study of *vv* embryos will add to our understanding of normal visceral torsion remains to be seen.

Literature Cited

1. Cockayne, E. A. *Quart. Journ. Med. N.S.* 7:479-494. 1938.
2. Gray, Peter. *Anat. Rec.* 82:416. 1942.
3. Lynn, W. Gardner. *J. Morph.* 79:1-26. 1946.
4. Wood, George O., and Alfred Blalock. *Archives of Surgery* 40:885-896. 1940.

Breeding Red Clover

THE utility of inbreeding in the improvement of various forage and pasture plants is an unresolved problem. In the study under review* Wexelsen is concerned with the question in relation to red clover (*Trifolium pratense* L.). The results of his extensive experiments not only fail to disclose an effective way to use inbreeding but also they lead to the conclusion that it should be avoided, for the most part, in working with this species.

A search for inbred lines which may be used as such for crop production is considered futile. Red clover is self-incompatible and, hence, exogamous. When inbred it displays the conventional features of a species replete with deleterious recessive inheritance. Sib matings for even one generation may drastically reduce seedling vigor, mature plant weight and seed production. Defective plants of several types come to light frequently. The amount of cross-incompatibility between individuals within families rises rapidly as inbreeding proceeds. The uniformity resulting from inbreeding is a dubious asset to a variety. It may even be disadvantageous. Adaptability to the varying conditions of soil and season would appear to be better assured by genetic diversity. Furthermore, plants demonstrably superior in breeding value to the average of the population from which they are derived occur very rarely.

The argument turns, therefore, to the value of inbreeding in the development of families for use in combinations. The justification for inbreeding in this relation must be that selection is made more effective thereby. Wexelsen points out that it has not been demonstrated that purging a variety of obviously unfavorable recessive characters raises the yielding capacity. Nor is productivity significantly increased by eradicating the conspicuously inferior individual plant components. It is held that in densely growing populations of red clover the weaker individuals are suppressed and yield per unit area is determined by the normal, vigorous individuals.

Selection between inbred lines of red clover proved effective in securing increased persistence; and, although Wexelsen was not directly concerned with the problem, he believes that inbreeding would be helpful in isolating disease resistant strains.

The probative value of outcrossing an inbred line intended for eventual use in combination with other inbred lines is emphasized. Limited tests by Wexelsen show that in red clover poor hybrids usually arise from poor inbreds. But the converse is not necessarily true; vigorous hybrids sometimes result from mating weak inbred families. The accurate assessment and more complete control of the cryptic heredity thus disclosed is the core of the red clover breeder's problem.

R. A. Brink
University of Wisconsin

*Wexelsen, Haakon: *Studies on Fertility, Inbreeding and Heterosis in Red Clover* (*Trifolium Pratense* L.). Dybwad, Oslo. 1945.

A BISEX STERILE VARIANT IN CUCURBITA PEPO

LAWRENCE C. CURTIS AND JOHN SCARCHUK
University of Connecticut, Storrs, Conn.

NORMAL AND STERILE ANTHERS
Figure 7

A SEARCH for sterile types of plants has been stimulated by the appearance of the brilliant study by Jones and his coworkers[2] which reports the successful utilization of genetic and cytoplasmically sterile types of onions to produce first generation hybrid onion seed for commercial planting. The possibility of using sterile types in other genera gives a new importance to these heretofore merely pathological types. In the genus *Cucurbita,* Hutchins[1] reported a male and female sterile line of *maxima* and Shifriss[3] has reported the occurrence of a male sterile line in the same species. This paper notes the occurrence of a male and female sterile line of *C. pepo.*

In 1947 a culture derived from three generations of selfing segregated for plants in which the anthers were rudimentary and contained no pollen (Figure 7). This culture, comprising 14 plants, contained four plants in which the male flowers opened and, from all external appearances, resembled normal flowers, except for the absence of anthers. The female flowers on these plants were also normal in external characteristics yet they failed to develop into mature fruit. All female blossoms began to rot within a week, regardless of whether they had been sib or cross pollinated.

Although the 10:4 ratio of normal to sterile plants closely approached the theoretically expected 3:1 ratio for a single pair of genes, a planting of remnant seed was made which produced 31 normal and 11 sterile plants. The results obtained in the original and replanted populations (41 normal, 15 sterile) closely approach the theoretical 3:1 ratio expected on the basis of a single factor difference.

Literature Cited

1. HUTCHINS, A. E. *Proc. Am. Soc. Hort Science* 44:494-495. 1944.
2. JONES, H. A., and A. E. CLARK. *Proc. Am. Soc. Hort. Science* 43:189-194. 1943.
3. SHIFRISS, OVED. *Jour. Hered.* 36:47-52. 1945.

PROVEN feeds for your "Lab" animals

FOR RABBITS

Globe Rabbit GLOB-ETS plus hay

Globe Rabbit Feed with alfalfa, pellet form — complete.

FOR RATS AND MICE

Dickinson's Dog Food — Nugget—Pellet—or Meal form

Dickinson's Kibbled Dog Food

Both of these feeds are dry —easy to handle

FOR GUINEA PIGS

Globe Rabbit GLOB-ETS plus hay

Globe Rabbit Feed with alfalfa, pellet form — complete

Supplement with Vitamin C carrier three times weekly

DICKINSON'S FEEDS for small animals are approved and used by many research laboratories. They are complete feeds, uniform, fresh, conveniently packaged—dry. Their usage by you will remove one variable in your research and experimentation. The Globe brand, produced by Dickinson, is backed by over 90 years of experience. Switch now to this reliable source for proven feeds for your experimentation animals.

FREE SAMPLES

If you would like to test Dickinson's Globe Feeds free, write us and we will gladly send a generous sample to you. Tell us how much and what kind—we will do the rest.

THE ALBERT DICKINSON COMPANY

2714 WEST 35TH STREET CHICAGO 32, ILLINOIS

Just Published

new 4th edition of
a standard textbook

HEREDITY

By A. FRANKLIN SHULL

Professor of Zoology, University of Michigan
McGraw-Hill Publications in the Zoological Sciences

355 pages, 6 x 9, illustrated. $4.00

As in previous editions, this well-known text presents the elementary principles of biological inheritance, with emphasis on those phases of the subject that are of special interest to mankind. The mechanism and behavior of genes and chromosomes are explained first and then the results of experiments in heredity are described. The new edition includes: gene action as indicated by the work of Beadle and others; the Rh factor in human blood, with practical applications; population and immigration problems; etc.

Send for a copy on approval

McGRAW-HILL BOOK COMPANY, Inc.

330 WEST 42ND STREET NEW YORK 18, N. Y.

An Introduction to Human Heredity and Eugenics:

HOW HEREDITY BUILDS OUR LIVES

BY ROBERT C. COOK
AND
BARBARA STODDARD BURKS

Illustrated with drawings by Clyde E. Keeler and photographs and charts from the *Journal of Heredity*. This booklet offers the student an informal introduction to heredity, environment, and their interactions.

64 pages; 27 figures; 75c per copy—$5.00 per dozen

AMERICAN GENETIC ASSOCIATION
1507 M St., N. W., Washington 5, D. C.

PLEASE

IF YOU ARE CHANGING YOUR ADDRESS, please let us know, if possible, four weeks in advance, giving old as well as new address, and adding zone number to meet postal requirements. If you are unable to notify us, so as to affect succeeding issues, kindly leave instructions and postage with your postmaster for forwarding your copy. Second class matter requires postage when remailed to a different address.

JOURNAL OF HEREDITY
1507 M Street, N. W.
Washington 5, D. C.

A Service for Those

TOO BUSY TO BROWSE

The **AGA Book Service** is designed for the convenience of the busy professional worker to whom books are not only the adjuncts of his pleasure, but, more importantly, the tools of his trade. The leisurely humanistic pleasure of browsing through the shelves of a well-stocked book store is all too frequently incompatible with the exigent schedule of the hard-pressed teacher, scholar or researcher. The facilities of the **AGA Book Service** provide the means, at a saving of time, money and trouble, of acquiring all the books you need.

- Just send us the title, author and publisher of each book
- • The books will be sent to you direct from the publisher.
- • • We bill you (at member's discount).

Order Your Books From

American Genetic Association Book Service
1507 M Street, N. W. **Washington 5, D. C.**